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PLANNING THE PROGRAM*

PRESIDENT'S ADDRESS

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AN Annual Meeting of the American Roentgen Ray Society is an enjoyable and educational experience. Through sixty years the present pattern has evolved and this pattern, with slight variations, has become stabilized so that there are four major areas of interest: Scientific Sessions, Scientific Exhibits, Instructional Courses and Technical Exhibits. There is no question that all of these are of value, perhaps even of equal value, but occasionally it seems worth while to stand aside and attempt an unbiased and unprejudiced appraisal of that part of the program with which one has been most intimately involved.

The chief duty of the President-Elect in conjunction with the Program Committee is to select scientific papers and arrange these for presentation at the Scientific Sessions. This is a task that is not without its difficulties and its hazards; hazards that confront every program chairman whether it be of a small local society, or a large and justifiably famous one such as the American Roentgen Ray Society. Planning for the scientific program of the present meeting began on the final day of the last one. An invitation was extended to all members of the society to contribute suggestions or abstracts, and letters were sent to many other individuals and centers where active work is being carried out. I am sure that the response varies from year to year, but in review of past programs it would seem that there is nearly always one "band wagon;" a popular area of study or investigation that may lead to a small deluge of papers. Sometimes one gets the impression that a paper is submitted not because of the intrinsic merit of the work but because of the extroverted personality of the author who just loves to be on any program. Papers of this type can be of great value to the program committee and are seriously considered, as, although the content of the talk may not be of the highest scientific caliber, the enthusiasm of the author often adds the needed sparkle to what may otherwise be a rather dull series of worthy papers. In addition, the committee is usually aware of a considerable amount of excellent work being done that should be presented to the Society but where there is undue reluctance exhibited

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by the physician. The serious investigator is often discouraged by the long gestational period of six or eight months that may be necessary before the paper can be presented at the Annual Meeting and then an equally long period before it can be published.

From these three groups, it is usually possible to collect forty or more papers that from their abstracts sound timely and interesting, but even with this accomplished any program committee is still worried. Is this enough? Will the audience be held? Or should we lard the program with individuals well known to radiology, who can be counted on to speak fluently and well on any subject? If by chance the content is equal to the delivery, one has indeed a prize addition to the program. These, "the aging stage army of the regular performers," certainly have a place on the program but it is not always clear just what that should be. Undoubtedly the Caldwell Lecturer, and I hope that the Lecturer of this Annual Meeting will forgive me for any reference to early aging, should be one whose scientific pursuits are deserving of the honor of the Society.

Your committee feels that the scientific program to be presented to you during the next four days is a worthy one. Possibly it could be better but I hope that any fault does not lie either with the speakers or with the committee itself. Many of the defects are wholly inherent in the size of the meeting. Discussions have been greatly reduced, as time is so limited. If a discussion ever has a value, it should be constructive criticism. But even destructive comments are of greater interest than the polite statements which seem to consist chiefly of, "I enjoyed the paper." The very few discussors who will appear on the program have been chosen because of their well-known ability to make acidulous comment in such a delightful way that no offense could possibly be taken.

It should be obvious to all that there is a need for smaller meetings, and for free give and take among the audience and the participants. For years the major societies in medicine have been burgeoning in attendance; many now take pride in the very number of registrants. Unfortunately, more registrants do not necessarily mean a better meeting. That some aspects of the meetings of the larger societies are not wholly satisfying is reflected by the tendency in the past few years for the formation of a number of small radiologic groups, perhaps international in scope and frequently limited to some particular segment of the broad field of radiology. These meetings, usually held on an informal basis, can have free discussion of completed studies or work in progress. Comment and criticism is invited. A prolonged gestation is unnecessary, and even an occasional abortion is expected, but the enthusiasm of the small audience is intense and the argument most rewarding. It is hoped that these scientific organizations will be kept small by firm and hard-minded admission committees because I believe that they have a very important role to play. Let the small group be the forum for the elaboration of ideas. A group selection can be made of that material or that paper worthy of presentation before a national audience. It should be possible to work out a table of organization where the best work presented at such Societies as the University Radiologists or the Pediatric Radiologists could be submitted to the scientific program committee of one of the major societies for consideration either as a scientific paper or as an exhibit. By some such mechanism it would seem possible to greatly strengthen all of the programs in bringing to them papers of proved merit.

I wish that a similar approach could be applied to the smaller groups of radiologists that meet in a city or district society throughout the year. Their tendency has been to follow the basic pattern laid down over the years, hesitating to attempt anything new for fear that it may not work. You all know the form which consists of an afternoon session of film reading which, sometimes, with an excellent moderator can prove of great educational value. How-

ever, all too often it is merely entertaining, since without spontaneity or a good moderator it may appear as a rather sad spectacle of reluctant participants discussing undiagnosable situations. It is of interest to note how high a percentage of these undiagnosable situations was by some unusual streak of brilliance diagnosed by one presenting the films. The late Dr. Merrill Sosman was one of the great moderators at this type of show. He always attributed to Seneca, and I hope correctly, the profound observation of, "The fact that you guessed and were correct is no excuse for having guessed in the first place." I hope that the film reading session will not be given up, but rather improved upon. Occasionally, the "film reading" is followed by a brief presentation by a local radiologist; even though his topic may be tremendously good, it is very rare indeed that he is allowed more than ten minutes. The speaker of the evening is usually a well-known figure of the radiologic scene making a sortie from his home ground who must deliver an address of almost exactly one hour in length. The young men of the society should have greater opportunity for participation and introduce to this forum material that later might be presented to a major society. These meetings can be interesting and educational if we will try for their continued improvement.

To be elected President of this or any other medical society is indeed an honor. To be made Chairman of the Program Committee is a great responsibility. This responsibility, coming as it does without warning or preparation, certainly tends to prevent radicalism or innovation in the planning of the program, but it seems that some changes and improvements are needed. These could well begin with the local societies. The specialty groups and radiologic hierarchies must at all costs avoid unwieldy size, too great formality of presentation and lack of free discussion. From these groups a constant and increasing flow should come of investigation worthy of presentation before a larger audience. If need be, the publication of the paper should not of necessity wait through the long period of printing of the program and printing of the journal. Perhaps through some such means, an even more interesting program than you will hear at this meeting will be available.

As I mentioned in the beginning, the Scientific Program is but one part of the educational experience provided by a meeting of the American Roentgen Ray Society. The Scientific Exhibits give an unexcelled opportunity for an essayist to present his material for a more careful study and afford an occasion for informal discussion, which of necessity must be lacking at the more formal presentation. The exhibit may be the only satisfactory way of reaching a competent audience, as often it is most difficult to present some material in a short essay so that it is thoroughly comprehensible. The Instructional Courses have nearly always been of an extremely high standard, even though rarely in the more remote past there has been a tendency to create a refresher course rather than have the instructor develop the advancing periphery of our knowledge. It hardly seems necessary to speak of the value of the Technical Exhibits. These for many years have been an integral part of the meeting, yet patronage has been avoided as a firm policy of the Society.

From these four facets of the Annual Meeting, one can acquire radiologic information of inestimable value. Along with this will go the pleasure of meeting again with colleagues, speaking for the first time to an author of some applauded work, the reunion of students with their former chiefs, the questions, the talk and the happy evening occasions that comprise this meeting. I trust they will be for you, as they always have been for me, a thoroughly enjoyable experience.

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A SAFE AND PRACTICAL INTRAVENOUS METHOD FOR ABDOMINAL AORTOGRAPHY, PERIPHERAL ARTERIOGRAPHY, AND CEREBRAL ANGIOGRAPHY*

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ANOTHER milestone in diagnosis of disease of the abdominal aorta, the peripheral arterial, and cerebrovascular circulation promises to be achieved by contrast roentgenography of these structures by the means of a safe and practical intravenous injection herein described. The method is a modification of the Robb and Steinberg technique for visualizing the cardiovascular system that has been available for over twenty years.[14,41,48]

The history of the development of angiography has recently been thoroughly reviewed by Kincaid and Davis[29] and so need not be repeated. Other publications deal with the various injection sites and techniques of angiography and stress the improvement brought about by rapid serial contrast roentgenography.[11,12] Egas Moniz in 1927[37] initiated cerebral arteriography. This has been a boon for diagnosis and treatment of cerebrovascular disease. Fortunately, complications resulting from arterial puncture and reaction to contrast substances have been uncommon, and even despite a rare fatality,[25] the data obtained are so important that it is worth the risks.[7] However, an intravenous technique for the visualization of the cerebrovascular circulation by lessening untoward reactions is obviously advantageous. Indeed, in 1956, Viallet and associates[39,52] reported such a method which consisted of the rapid single injection of concentrated contrast material and the making of serial studies of the total cerebrovascular circulation.

Translumbar aortography was introduced by dos Santos *et al.*[15,16] in 1929. Excellent visualization of the abdominal aorta was achieved from the very beginning, but many years elapsed before the translumbar method of injection won general acceptance.[29] Of late, rare instances of fatalities and uncommon but serious complications have been recorded in the literature[1–4,6,8,9,16,24,26–28,30,31,34–36,43,49,50] This has resulted in understandable reluctance, largely because of legal implications, to use this method of aortography. Accordingly, a new safe and practical intravenous method of abdominal aortography, peripheral arteriography, and cerebral angiography is of considerable clinical significance.

Early, during development of the intravenous method of angiocardiography,[14,41,42,44] opacification of the abdominal aorta was often secured. This was especially true in infants and children because their small stature frequently permitted the visualization of the abdominal aorta with left heart opacification. Later, when a technique for nephrography was developed, the abdominal aorta was occasionally seen,[40,51,53,54] but this never occurred as frequently as with nephrotomography.[17,18,19] Utilizing serial roentgenography the abdominal aorta was often readily visualized.[42] Recently, Bernstein and associates[5] made a preliminary report of abdominal aortography by the intravenous route. A mechanical injector, timing of the opacification of the abdominal aorta with radioisotopes, application of peripheral vascular occlusion and elaborate roentgen equipment make the procedure formidable and expensive.

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METHOD

The intravenous method of abdominal aortography and peripheral arteriography depends upon the principles developed for angiocardiography.[14,41,47,48] Speed of injection of the contrast material is essential and this is accomplished by simultaneously injecting the large veins of both arms. This eliminates the factor of dilution of the contrast material by blood from the opposite innominate vein when only one injection is made and, of course, increases the bolus effect of the contrast material into the circulation. Speed of injection is also facilitated by elevating the arms and having the patient perform respiratory maneuvers.

The time of roentgen exposure of the abdominal aortic or peripheral arteriogram is determined by the preliminary circulation time with sodium dehydrocholate (decholin). Standard roentgen technique similar to that of intravenous pyelography, which makes use of the Bucky grid, is all that is needed for roentgenography of the abdominal aorta and its branches. No special roentgenographic equipment is required.

The percutaneous insertion of the 12 gauge Robb-Steinberg needle stop-cock unit has previously been described.[14,41] In the absence of a large vein, it may be necessary to perform a cut-down for insertion of the needle. The position of the cannula is then secured by adhesive tape (Fig. 1*A*). To make certain that the cannula is properly seated in the vein, an injection of normal saline to safeguard against extravasation is advisable. The patient is then briefed about the technique of the examination and is instructed about breathing during the examination.

The determination of the preliminary circulation time with the patient supine will serve as a good rehearsal of the patient's participation in the test. Sodium dehydrocholate (decholin), 3 cc., is mixed with 15 cc. saline in a 50 cc. syringe and attached to one of the cannulas. The arm

Fig. 1. *The technique of rapid, bilateral, simultaneous, intravenous abdominal aortography, peripheral arteriography and cerebral angiography.* (*A*) The patient is supine on a regular roentgen table containing Bucky grid carriage and 14×17 inch cassette (C) placed under abdomen for visualizing the abdominal aorta. Note the Robb-Steinberg cannulas (arrows) inserted into cubital veins and secured by adhesive tape. (*B*) The Robb-Steinberg syringes containing the contrast medium are attached to the cannulas (arrows) and simultaneous injections are made. The physicians, clothed in protective gowns, are standing on a 12 inch platform. Note the elevation of the arms and alignment of syringes and needles to course of the veins. The roentgen tube (T) is in position for exposure of the abdomen at a predetermined interval after the beginning of injection.

is then elevated, making certain that it is relaxed at the shoulder (this avoids physiologic obstruction of the subclavian vein underneath the first rib). The stop-cock is then opened and the patient is instructed to "breathe out" gently. Then he is asked to "breathe in" making certain that only a moderate breath is taken, for it is important to avoid the Valsalva maneuver which tends to delay circulation into the heart. Injection of the decholin-saline mixture is made simultaneously, with the direction to "breathe in." This also is the signal for the assistant to start the stop-watch. Once the injection is completed, the patient, having been told to anticipate a bitter taste, is urged to go through the motion of tasting by smacking the lips, tongue and mouth. This automatically releases the tendency to perform the Valsalva maneuver. The onset of the bitter taste is then recorded by stop-watch. The modified circulation time from arm to tongue is used as a guide for the starting time of the roentgen exposure for the abdominal aorta. If the accuracy of this determination is uncertain, a repeat circulation time determination is done. The exposure for abdominal aortography is prolonged to two or three seconds and begins about one-half second after the circulation time.

The patient is told to anticipate an intense wave of heat soon after injection of the contrast medium and assured that it will quickly pass. All is then ready for the aortographic study. The special Robb-Steinberg syringes are loaded with concentrated iodide contrast material, a mixture of the (30 per cent) sodium salt and the 60 per cent n-methylglucamine salt of 3,5-diacetamido-2,4,6-triiodobenzoic acid (hypaque 90 per cent or cardiografin), the amount depending on the weight of the patient. A dose of 1 cc. per kilogram of body weight divided equally for injection into each arm is advised (Table 1). Up to 50 cc. in each arm may be necessary for obese patients over 200 pounds. The syringes are then attached to the cannulas, the stop-cock opened, and the arms elevated with a

TABLE I

CONTRAST MATERIAL DOSAGE CHART FOR INTRAVENOUS ABDOMINAL AORTOGRAPHY, PERIPHERAL ARTERIOGRAPHY AND CEREBRAL ANGIOGRAPHY

Weight in Pounds	Kilograms*	Dose in each syringe (cc.)	Total Dose (cc.)
100	50	25	50
150	70	35	70
180	80	40	80
200	90	50†	100

* Approximate weight.
† Maximal dosage advised although 45 cc. per syringe may be adequate for this weight.

physician standing on a platform on each side of the patient (Fig. 1*B*). At the signal, "breathe in," the technician starts the stop-watch. The simultaneous injections are made rapidly and completed in one and one-half to two seconds. Because of viscidity the concentrated contrast agent may be difficult to inject rapidly unless the mixture is warm. The patient is then told to "breathe naturally." Two or three seconds prior to the selected exposure time, the patient is ordered to "stop breathing." A two second roentgen exposure for abdominal aortoggraphy is then made while the patient stops breathing. If peripheral arteriography is desired, another exposure may be made either with a portable roentgen apparatus or with another tube. Standard grid cassette technique for lower extremity roentgenography suffices. If, after inspection, the roentgenogram proves satisfactory, the cannulas may be removed and a tight temporary bandage applied. There is no hesitancy in repeating the injection after a short interval if the patient has tolerated the procedure. Also, if the peripheral circulation at the bifurcation of the iliac arteries is desired, the Bucky grid carriage may be shifted to include the extremities and another injection made.

RESULTS

The first successful abdominal aortogram, with rapid, bilateral, simultaneous

intravenous injection of concentrated contrast material and prediction of abdominal opacification, was made on March 31, 1959 (Fig. 2). Twelve additional patients have been studied since then and in each instance the abdominal aorta and its branches were visualized. In one case the cerebral circulation was recorded by means of the F-X-R magazine.[45] In several other instances peripheral arteriography by exposure of films under the legs with a portable roentgen apparatus was obtained. The normal abdominal aorta is shown in Figure 3*A* and the normal peripheral arterial circulation of the same patient is demonstrated in Figure 3*B*. The normal peripheral arterial circulation of another patient made with a grid cassette under the pelvis and upper femoral region is seen in Figure 4.

Arteriosclerosis of the abdominal aorta was revealed in another patient (Fig. 5*A*); the femoral arterial circulation, especially the left, showed marked arteriosclerotic involvement (Fig. 5*B*). Another example of advanced arteriosclerotic disease of an extremity was seen when the peripheral circulation was visualized during examination of the abdominal aorta of a woman suspected of having an aneurysm (Fig. 6).

Abdominal aortography via intravenous injection disclosed aneurysms in 4 patients. In one, a splenic artery aneurysm was suspected because of calcifications in the left upper quadrant (Fig. 7*A*) and a splenic artery aneurysm was visualized (Fig. 7*B*). In another patient, a pulsating abdominal mass and faint linear calcification along the right side of the spine were manifestations of an abdominal aortic aneurysm (Fig. 8*A*). Confirmation by intravenous abdominal aortography was had, although two injections were required to visualize the abdominal aorta completely (Fig. 8, *B* and *C*). The excised specimen (Fig. 8*D*) is included to substantiate the roentgen findings. A third patient had clear-cut evidence of a huge abdominal aneurysm (Fig. 9); an attempt to secure more complete opacification of the aneurysm by making another injection was a complete failure—the only one in the series. Fortunately, the earlier study (Fig. 9) was satisfactory.

Fig. 2. *The first intravenous abdominal aortogram with bilateral simultaneous injection of contrast material made on March 31, 1959.* The entire abdominal aorta and branches are well opacified. The patient was a seventy-one year old man suspected of having an aneurysm. Slight irregularity of the abdominal aorta and "buckling" of the iliac arteries and branches are characteristic of arteriosclerosis.

Technique: The patient weighed 60 kg.; the modified preliminary circulation time (decholin) was ten seconds. Thirty-five cc. hypaque (90 per cent) was injected into each arm at a speed of two seconds. Thickness of abdomen was 20 cm.; distance of tube to table top, 44 inches; milliamperage, 200; kilovoltage, 58; and the exposure was started at ten and one-half seconds after the beginning of the injection and continued for a duration of two seconds. The reaction was mild and transient.

A patient with frank aortic regurgitation, hypertension and cardiac enlargement (Fig. 10*A*) also had an abdominal aneurysm and marked arteriosclerosis of the leg vessels (Fig. 10*C*). Visualization of the abdominal aneurysm was undertaken to determine if the femoral arteries were sufficiently patent to allow blood flow, if the

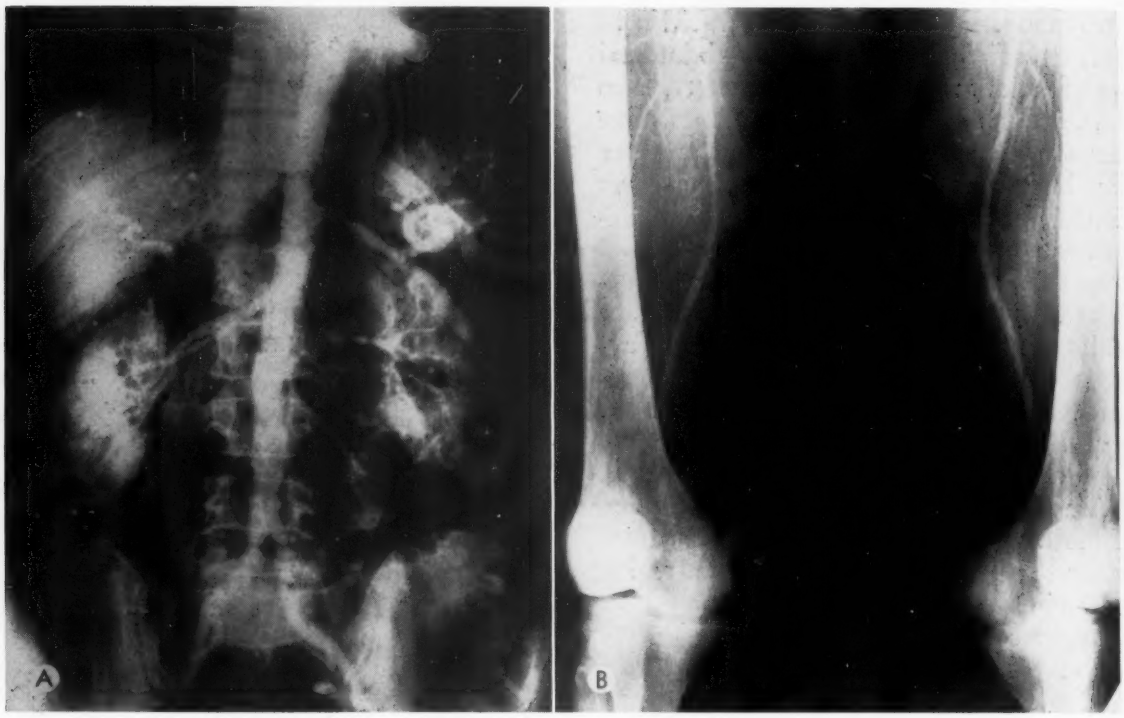

FIG. 3. *Intravenous normal abdominal aortogram and peripheral arteriogram.* (*A*) A fifty year old man suspected of having abdominal and peripheral vascular disease was found to have a normal abdominal aorta and branches. (*B*) The normal peripheral arterial circulation.

Technique: Weight 75 kg.; preliminary circulation time ten seconds; 50 cc. contrast agent was injected into each arm; the reaction was mild and transient. The peripheral arteriogram was made with a portable roentgen apparatus using a grid cassette. Distance, 48 inches; milliamperage, 30; kilovoltage, 50; and exposure time 2.0 seconds.

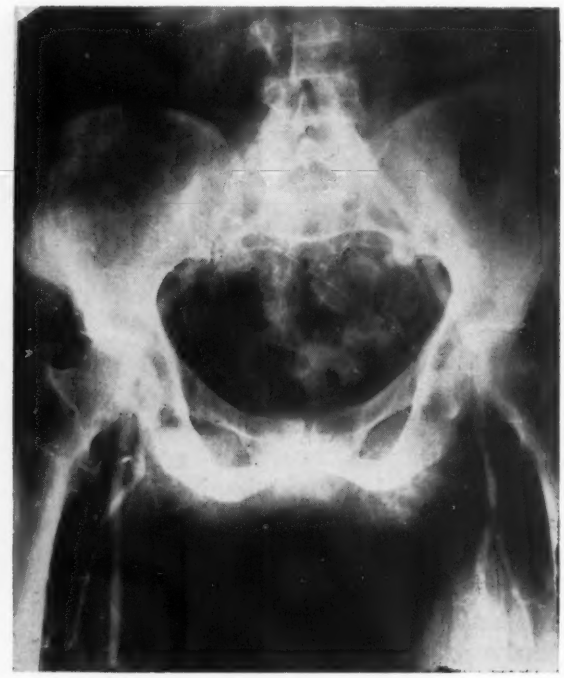

FIG. 4. *Intravenous peripheral arteriogram showing the normal iliac arterial tree.* A hypertensive fifty-five year old woman with buckling of the innominate and carotid arteries had a grid cassette placed under pelvis during cerebral angiography by intravenous injection and a roentgenogram of the iliac arterial circulation was obtained.

Technique: Weight, 78 kg.; preliminary circulation time ten seconds; 50 cc. contrast agent was injected simultaneously into each arm; the reaction was mild and transient. The F-X-R magazine, at one second intervals, was used beginning at eight seconds for cerebral angiography. The lower abdominal arteriogram was made with a grid cassette at eleven seconds after the beginning of injection; the duration of the exposure was one second.

Fig. 5. *Intravenous abdominal aortogram showing advanced abdominal aortic and peripheral vascular arteriosclerosis.* (*A*) A fifty-two year old woman with claudication of the legs had marked narrowing of the aorta just below the renal arteries and above the common iliacs (arrows) with dilatation of the aorta between these areas. (*B*) The femoral arterial circulation was markedly diminished, especially on the left side; R and L are the right and left legs.

aneurysm were resected and replaced by a graft. Despite a delayed modified circulation time of twenty seconds (twice the normal determination), a successful aortogram resulted via intravenous injection and a huge thrombosed aneurysm was demonstrated (Fig. 10*B*). The peripheral arterial circulation, though markedly arteriosclerotic, was patent (Fig. 10*D*).

DISCUSSION

The advantages of securing abdominal aortography, peripheral arteriography and cerebral angiography by intravenous injection are obvious. No longer need there be hesitancy in attempting to visualize these structures since the complications of arterial puncture, especially those of translumbar aortography, are entirely avoided. Now that surgical excision of aneurysms and replacement by homografts and synthetic materials are possible, aneurysmal and occlusive disease of the abdominal aorta and its branches is no longer of academic interest. With elimination of the necessity for anesthesia and the hazards of direct abdominal aortic puncture, greater use of intravenous aortography for diagnosis of disease of this system will be forthcoming. Furthermore, the benefits and results of surgery can be readily demonstrated and the complications and shortcomings of surgical procedures may also be evaluated.

Since the principles of visualizing the thoracic aorta angiocardiographically are similar to those which apply to the abdominal aorta, it is important to pay close attention to the details of the method of angiocardiography.[14,41,47] While retrograde aortography may be indicated for coronary arteriography, the differentiation between a patent ductus arteriosus and an aorticopulmonary window, and in the differential

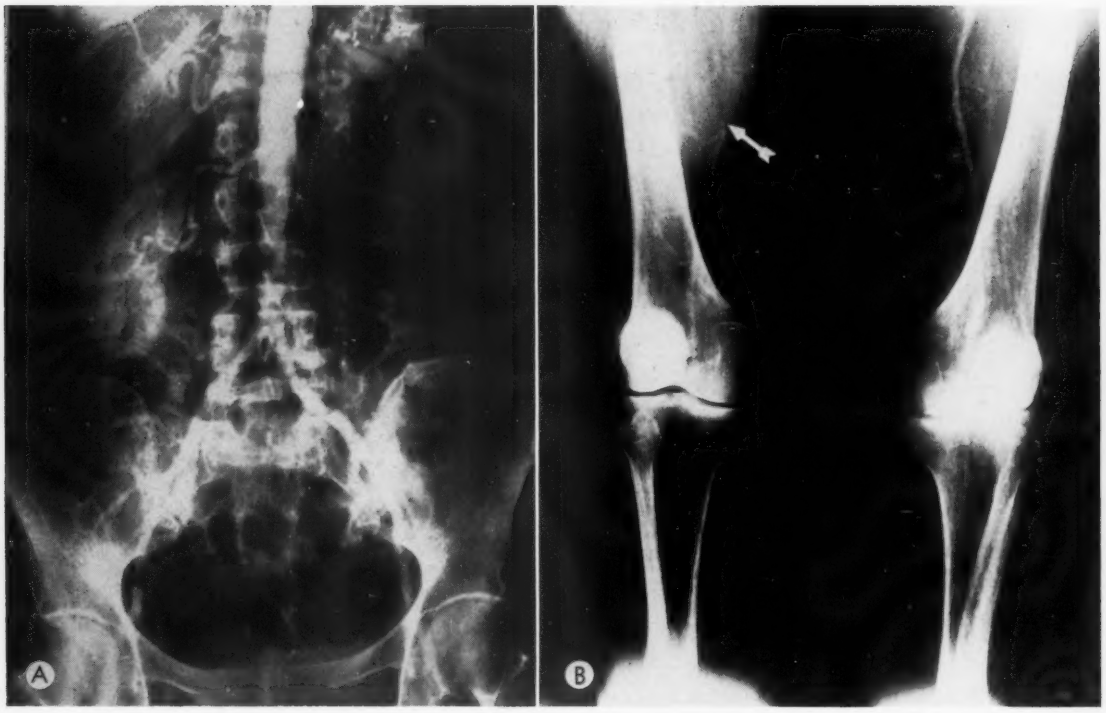

FIG. 6. *Intravenous peripheral arteriogram showing peripheral vascular disease in a woman suspected of having an abdominal aortic aneurysm.* A fifty-nine year old woman with a previous gastric resection had a palpable pulsatile abdominal mass. (*A*) Abdominal aortography disclosed an intact, although narrowed aorta. The metallic clips along the left side of the spine are the sites of the previous abdominal surgery sutures. (*B*) There is advanced arteriosclerosis of the femoral arteries, especially on the right side where there is an absence of opacification of the femoral artery (arrow) with many collaterals.

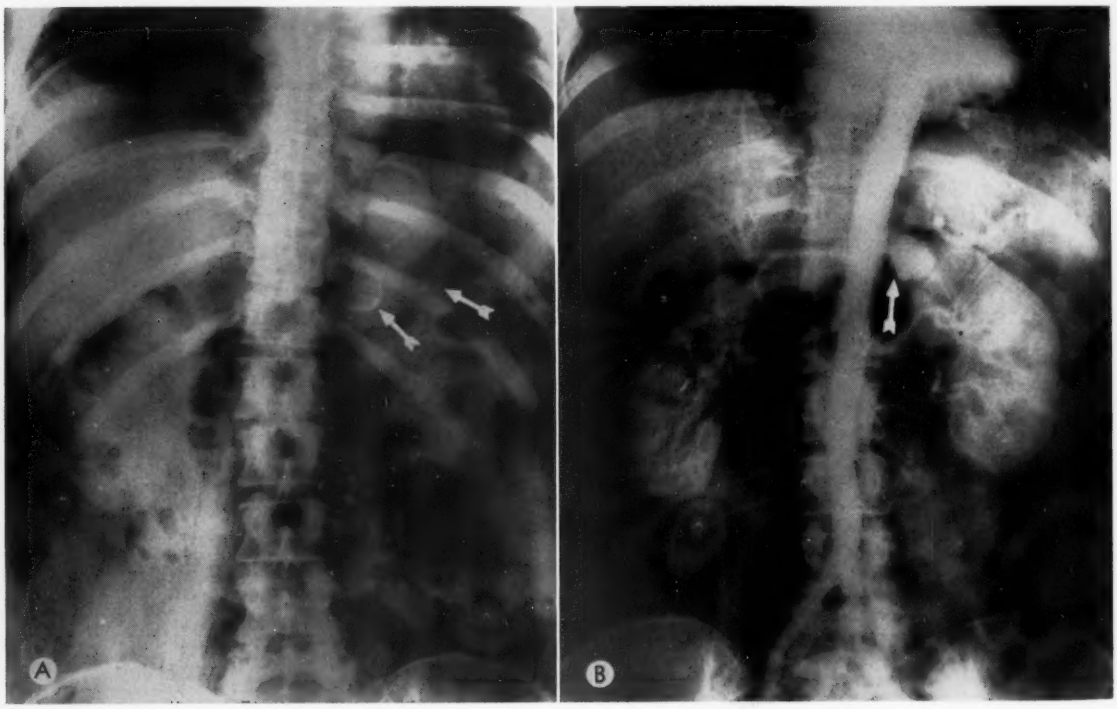

FIG. 7. *Intravenous abdominal aortogram showing a saccular aneurysm of the splenic artery.* (*A*) A fifty-three year old woman had multiple calcifications (arrows) in the left upper quadrant of the abdomen. (*B*) Intravenous abdominal aortography revealed a 2.5 cm. rounded aneurysm of the splenic artery (arrow). The more laterally placed calcific nodule was not opacified and was found to be a calcified mesenteric lymph node at operation.

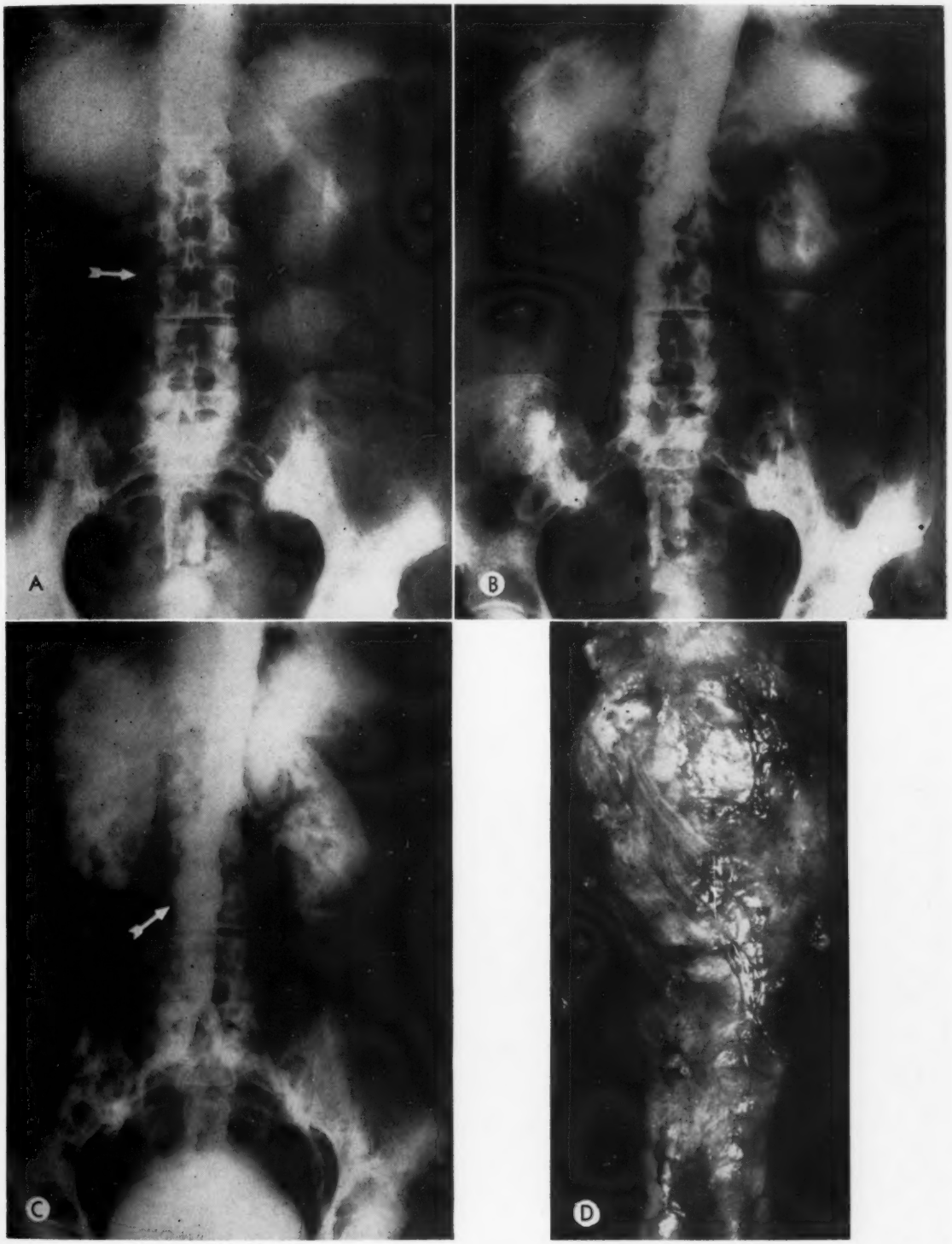

Fig. 8. *Intravenous aortogram showing an abdominal aortic aneurysm.* Later the aneurysm was resected and replaced by teflon prosthesis. (*A*) The conventional roentgenogram of a sixty-five year old woman with a pulsatile abdominal mass and linear calcification adjacent to the right border of the third and fourth lumbar vertebrae (arrow). (*B*)Intravenous abdominal aortogram, made eleven to thirteen seconds after the beginning of injection (decholin circulation time, 12 seconds), showed only partial opacification of an abdominal aortic aneurysm. (*C*) Repeat study, made thirteen to fifteen seconds after injection, opacified the entire abdominal aorta and aneurysm (arrow). (*D*) The gross specimen following resection of the aneurysm.

FIG. 9. *Intravenous abdominal aortogram showing a huge aneurysm of the abdominal aorta.* A fifty-six year old man with a large pulsatile abdominal mass was found to have a 6 by 10 cm. aneurysm of the abdominal aorta beginning below the renal arteries (arrow). Resection and replacement prosthesis could not be done because of involvement of renal arteries.

diagnosis of a ruptured aortic sinus and a coronary arteriovenous fistula, it need not be used for routine visualization of the aorta.[47] Failure to opacify the thoracic aorta angiocardiographically is usually due to disregard of the principles of speed of injection, proper positioning of the patient and attention to roentgenographic details.[14,41]

The concentrated contrast materials now available commercially are well tolerated intravenously even in large doses.[13,21] Indeed, Lemmon and associates[33] and Finby and Goldsmith[23] have injected 30 to 60 cc. of highly concentrated mixtures of sodium and methylglucamine salts of diatrizoate directly into the ascending aorta for coronary arteriography and thoracic aortography and noted no ill effects. For ventriculography[10,32,38] as much as 30–50 cc. of highly concentrated organic iodide solutions have also been injected directly into the heart chambers. Since the large doses of contrast substance employed in our method are given intravenously, it is not surprising that they are well tolerated. It is our opinion at this time that the dosage of concentrated contrast material should be 1 cc. per kilogram of body weight. Eleven patients had mild and transient reactions to injection of the contrast agent, consisting of intense heat and flush, although the dosage now recommended was exceeded in the early trial period. Two patients, both women, had moderately severe reactions consisting of overwhelming heat and momentary apprehension associated with a "sinking feeling." The pulse of one of the patients during the moderately severe reaction became faint for three minutes; in the other it remained regular and of good quality. The heart rate, too, was rapid but regular; the sounds were of good quality. Blood pressure determinations are difficult because of the bilateral cannulas. In 2 patients the blood pressure was at the preinjection level when it was secured, soon after the removal of the cannulas. Strict adherence to the dosage schedule (Table 1) is recommended in order to avoid severe reactions.

Thrombophlebitis has not proved to be troublesome. In several instances double injections have been tolerated without apparent venous damage. The usual reaction to the simultaneous double dose is mild and transient and very much like that in angiocardiography with a single injection, except that there is a more intense flush and a greater sensation of heat, traveling from head to toe. The reaction is probably also aggravated by the supine position, since reactions are usually less pronounced in erect patients during angiocardiography. Vomiting does not occur when the study is made during the fasting state (the meal prior to the examination is omitted). Reactions to the contrast material can be markedly diminished if there is a calm, reassured, nonapprehensive attitude.[22] In contrast to translumbar aortography, where there is danger to the kidney when the

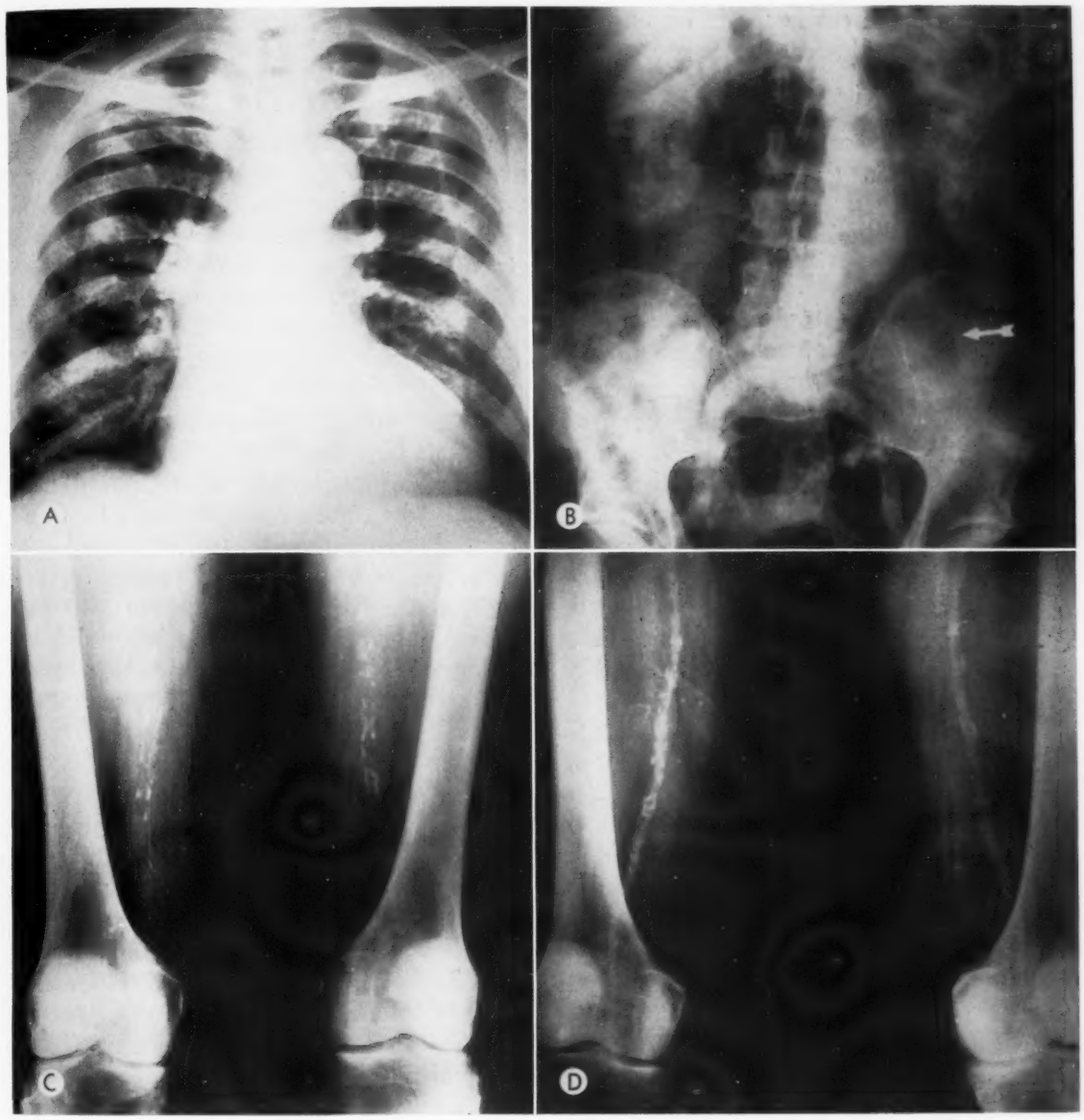

Fig. 10. *Intravenous aortography and peripheral arteriography in a sixty-three year old man with aortic regurgitation, aneurysm of the abdominal aorta, and arteriosclerosis of the peripheral arteries.* (*A*) Teleroentgenogram of the chest revealed a huge left ventricle and widened ascending aorta (the blood pressure was 240/110 mm. Hg). (*B*) Intravenous abdominal aortogram showed a sacciform aneurysm with a huge laminated thrombus demarcated by linear calcifications (arrow). (*C*) Conventional roentgenogram of the lower extremities revealed marked calcification of the femoral arteries (arteriosclerosis). (*D*) Intravenous peripheral arteriography demonstrated opacification of the femoral arteries, which contain calcifications in the vessel walls.

aorta is occluded by thrombus, visualization of the renal arteries via the intravenous routes does not seem to harm the kidneys. The urine and the blood urea nitrogen on two successive days in 2 patients were normal.

The roentgen exposure time of the abdominal roentgenogram has been two seconds. In contrast to cardiac chamber and great vessel visualization which requires short exposure times,[14] the abdominal aortic and peripheral arterial exposure times may be prolonged. The present technique results in only one contrast roentgenogram of the abdominal aorta. A simultaneous angiogram of the peripheral ar-

terial tree can be easily made by another exposure of the lower extremities (with suitable coning to prevent fogging of the abdominal roentgenogram) with a portable roentgen unit. Since only three or, at the most, four films (including preliminary positioning and pyelographic roentgenograms) are exposed during a contrast study of the abdominal aorta, the amount of radiation to the patient is low. The physicians making the injection receive very little exposure if the roentgen beam is suitably collimated and conventional protective garments are worn. As a further precaution, the arm, once the injection is completed, may be placed at the side of the patient on the table and the operators can move to a shielded area. Sufficient time for this is afforded because the exposure of the roentgenogram is made ten seconds or more after the beginning of injection; the injection is usually concluded at the end of two seconds.

The personnel for performance of abdominal arteriography via the intravenous route consists of two physicians who make the injections and a technician. This is much less formidable than translumbar aortography, where the injector must maintain sterile precautions necessitating a trained attendant. If general anesthesia is required for the translumbar study, another trained nurse or anesthesiologist is needed. Since local anesthesia is used only for insertion of the cannulas for the intravenous method, general anesthesia is not needed.

Determination of the preliminary modified circulation time with decholin prior to the injection of the contrast material is advised for every patient. This not only indicates whether there is obstruction (physiologic or pathologic) to blood flow into the heart but may also predict inability to secure a satisfactory study. Delay of passage of the contrast material occurs in cases of pulmonary disease associated with cor pulmonale, some types of congenital heart disease (Ebstein's anomaly, aortic stenosis), congestive heart failure and valvular disease such as mitral and aortic stenosis or regurgitation. It is possible that in such instances abdominal aortography may not be successful because of dilution of the contrast material and disruption of its continuity. Fortunately, patients requiring abdominal arteriography are usually free of congenital heart disease, congestive heart failure and valvular heart disease. Two patients in our small series with short delays in the circulation time due to chronic lung disease and mild pulmonary hypertension had excellent opacification of the abdominal aorta (Fig. 4 and 8, *B* and *C*). Another patient with frank aortic regurgitation and a long circulation time also had good opacification of an abdominal aneurysm (Fig. 10*B*).

Further refinements (all expensive) include serial roentgenography of the abdominal aorta and its branches. Biplane angiography will also provide a simultaneous lateral view, especially valuable in the study of cerebral angiography. Injection into both arms with the patient on the side for a lateral study alone may prove difficult and require mechanical injectors. Also, in such instances, percutaneous insertion of the cannula cannot be used because the high pressures will dislodge the cannula and result in infiltration. Improvement in organic contrast agents, especially a higher iodine content, providing greater opacification and, of course, less reactivity will appreciably reduce the quantity of material needed for injection. This may make it possible to routinely visualize the abdominal aorta and branches with unilateral rapid intravenous injection.

Contraindications to intravenous abdominal aortography and cerebral angiography are rare. The patient should be cooperative and well enough to lie on a roentgen table for at least one-half hour. Unconscious or anesthetized patients may, however, be examined successfully; experience with angiocardiography indicates that anesthetized patients may be injected rapidly without the aid of respiratory maneuvers. In such instances an objective

type of circulation time may have to be used in order to estimate the time of exposure of the abdominal and cerebral roentgenograms. No longer need azotemia contraindicate study of the circulatory system using organic iodides. Indeed, such compounds are used widely for diagnosis of kidney disease even when uremia is present. Diagnosis of an obstructive urologic lesion amenable to surgical alleviation or cure far outweighs the risk, if any, of adding to nitrogenous wastes in the blood. Furthermore, in the presence of poor renal function, much of the contrast material is excreted in the biliary tract.

The increasing use of splenoportography without harm in patients with cirrhosis of the liver also demonstrates that advanced liver disease is not a contraindication to the injection of the organic iodide compounds into venous channels.[20] Since the organic contrast compounds are securely bound to the iodide molecule, patients allergic to the iodides need not be deprived of diagnostic studies with such agents. In our large experience many allergic patients have tolerated angiocardiographic and urographic studies without ill effects.[22] Finally, there remains the rare individual who reacts severely to the radiopaque material in an anaphylactic fashion. Since preliminary testing, whether intradermal, ocular, oral or intravenous, does not designate the severe reactor, routine pre-testing or premedication need not be done. If there is a clear indication for study of the circulatory system, the rare fatality must be accepted as an unavoidable risk. However, the availability of emergency treatment for severe reactors will further cut the risk.[22] Good clinical judgment based on experience is all important for selection of the patient suitable for study; this will also detect the high-risk patient who requires special facilities for the intravenous injection.

More studies of patients and confirmation by others are needed to thoroughly test the validity of the method of intravenous abdominal arteriography, peripheral arteriography and cerebral angiography described above. The uniform and entirely successful visualization of the abdominal aorta in 13 consecutive patients—even though 2 injections were required in one case for complete visualization of an abdominal aortic aneurysm (Fig. 8*D*)—and the sound principles tested during the twenty year period of angiocardiography make it certain that intravenous aortography, peripheral arteriography and cerebral angiography will become a valuable diagnostic method. Bilateral filling of the carotid and vertebral systems that occurs following intravenous injection should not handicap interpretation of cerebral angiograms, once the normal totally opacified vascular anatomy of the cerebral system is appreciated.[39,52] Simultaneous bilateral injection of the saphenous or femoral veins also provides a superb inferior vena cavagram. Subsequent serial exposure may also show opacification of the portal venous system (according to a single observation). Intravenous abdominal arteriography should also prove to be valuable for diagnosis of renal arterial anomalies and diseases causing hypertension, especially those amenable to surgery. Since the liver was unusually well opacified in 2 patients who had had a double injection for abdominal aortography (each with a total dose of 150 cc. contrast material), this method also enables hepatography. Serialographic study of the liver in one of the cases revealed the entire contour of the liver, providing an even more detailed study. Accordingly, body section roentgenography during the hepatogram phase may disclose congenital malformations, liver abscesses and tumors (primary and metastatic). A precedent for this is nephrotomography which was developed in this center.[17,18,19] Of course, nephrotomography may be done after visualization of the abdominal aorta. Because of the larger dose of contrast agent, a striking study results.

Complete opacification of the circulatory system in man is now feasible. To accomplish this ideally, biplane angiocardiography at onset of the intravenous injec-

tion will provide studies of the entire cardiovascular system. Cerebral angiography, preferably biplane, may then be secured beginning with the systemic (decholin) circulation time. Soon after (one-half to one second), roentgen exposure of the abdomen will visualize the abdominal aorta. Finally, the peripheral circulation (the exposure time commencing at the conclusion of the abdominal aorta study) can be visualized. Motion of the various circulatory components may also be had by cineangiography.

Although a total survey of the circulatory system may be of limited clinical value, the data may prove invaluable for physiologic study. Continuous electrocardiography during intravenous injection will relate the opacification of the various systems to the cardiac cycle. Biplane studies will permit volumetric determinations of the cardiac chambers, great vessels and cardiac output. Variations of the circulatory system with systole and diastole and propagation of the pulse can also be evaluated.

New syndromes due to generalized and localized delay of blood flow in the cerebrovascular system which were discovered when cerebral angiography via intravenous injection was performed have already been described.[39] Further studies may disclose new and previously unknown circulatory patterns which may be of diagnostic value in the various types of mental deficiencies.

SUMMARY AND CONCLUSIONS

A safe and practical intravenous method of abdominal aortography, peripheral arteriography, and cerebral angiography, making use of physiologic principles learned during angiocardiography, is described. This method has distinct advantages over the translumbar, carotid and vertebral arterial injection routes which have sometimes been associated with serious complications and occasionally death.

Rapid and simultaneous injection of concentrated organic iodides can be made by injection into both arms with special syringes attached to Robb-Steinberg cannulas. If dosages of 1 cc. per kilogram of body weight are used, the reaction to injection of large quantities of the contrast material will be well tolerated. Since conventional roentgen equipment is all that is required for the intravenous method of abdominal aortography and peripheral arteriography, the study can be performed in almost every radiologic department.

Aneurysmal and occlusive disease of the abdominal aorta and branches can be readily diagnosed, facilitating the appropriate treatment for each patient. Cerebral angiography via the intravenous route, especially when biplane studies are made, will open a new field of diagnosis. Vascular malformations (aneurysms) and displacement of cerebral structures may furnish clues to hitherto unrecognized syndromes of brain disease. Angiographic studies to locate subdural and cerebral hematomas following cerebrovascular accidents may well become routine.

Finally, modification of the Robb-Steinberg method of intravenous injection of concentrated contrast material, by making a double simultaneous injection, renders possible the visualization not only of the central cardiovascular system (the great veins, the chambers of the heart, the pulmonary circulation and the thoracic aorta), but also of the abdominal aorta, peripheral arteries and cerebral vessels. Thus, total opacification of the circulatory system is achieved. An alternative method of visualization of the portal system and laminagraphic study of the opacified liver (hepatotomography) require further investigation.

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ADDENDUM

Since this paper was submitted for publication on May 6, 1959, this method was success-

fully employed in additional cases, raising the total number to 52. The abdominal aorta and peripheral arterial circulation were visualized in every case.

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SIMULTANEOUS BIPLANE LARGE-FILM SERIALOGRAPHY IN CEREBRAL ANGIOGRAPHY*

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SINCE the introduction of cerebral angiography in 1928,[3,4] there has been a continuing search for methods of optimum serialographic examination of the cranial circulation. A number of serialographic units have been introduced.[1,2,5–16] Some of these have proved quite effective. However, the search has continued for improved technical aids in this area and the need for biplane visualization has been obvious from the very beginning.

It is the purpose of this report to describe our utilization of a biplane, large-film, serialographic unit which was originally purchased for selective catheter angiocardiography but also has been adapted to cerebral angiography with effective results. We have been encouraged to report our results with this unit inasmuch as a number of well-informed radiologists from other cities visiting our department have evinced great interest and some surprise at the use of this biplane film changer in cerebral angiography. We are, therefore, presenting a brief description of the application of this biplane film changer to cerebral angiography for the benefit of those who similarly may wish to take advantage of such a unit, if available, or if its purchase is contemplated.

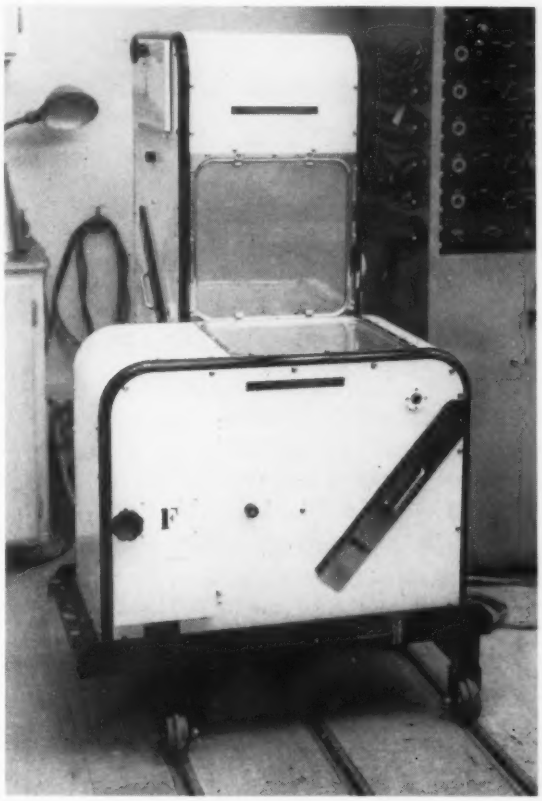

FIG. 1. The biplane film changer described in the text is illustrated.

METHOD

The biplane film-changing device that we are using is manufactured by the Elema-Schönander Corporation (Fig. 1). This unit includes two 500 ma. generators and uses 14×14 inch cut film. These film changers permit up to 30 exposures in each of the two planes simultaneously. Each sequence may be run as slowly as three films per two seconds or as rapidly as six films per second. For cerebral angiography the patient is placed supine on a special sliding table which lies on the Bucky table with the anteroposterior beam directed downward and the lateral beam directed across the table. A thirty-six inch target-film distance is used in each of the two planes. In the sagittal plane, the back of the head is positioned three inches above the anteroposterior film changer and, in the lateral plane, the lateral aspect of the patient's head is placed 4 to 5 inches from the lateral film changer (Fig. 2). In the average case, the factors used are 86 kv. and 20 ma. seconds. Each film changer has an 8:1 specially de-

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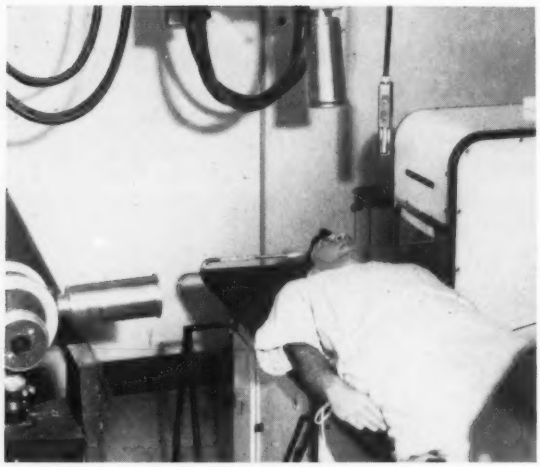

FIG. 2. The biplane film changer is seen in place with the patient positioned on the sliding-top table.

signed fixed focus grid and standard 4 inch circular extension cones are employed. Cross-hatched grids are not necessary. Standard film is used and high speed screens are preferable. Magnification occurs in the order of approximately 10 per cent in the anteroposterior plane and 15 per cent in the lateral plane.

In the usual cerebral angiographic examination, a speed of three films per two seconds carried through for a total period of nine seconds is generally sufficient. A total of ten films in each of the two planes is thus obtained. Currently, a hand syringe is used for the percutaneous injection by the neurosurgeons but we are now investigating the feasibility of employing the matching high-pressure automatic syringe (Gidlund) (Fig. 3) which is utilized in selective catheter angiocardiography. This automatic injector which uses compressed air and a stainless steel syringe has certain helpful features: (1) the opaque solutions can be pre-warmed and kept at constant temperature in the syringe; (2) the amount of air pressure is pre-set and varied as needed; (3) the volume of medium used can be pre-set; (4) the tripping of the generator is automatically set off at a predetermined time during any phase of the injection; and (5) the operator avoids all radiation exposure since he does not remain in the room.

The simultaneous biplane examination usually requires only one injection of opaque medium but, rarely, oblique views or a submentovertex projection may be found advisable and thus a second injection may be necessary.

ADVANTAGES

The advantages inherent in the use of this biplane unit are:

1. Scout roentgenograms are readily obtained prior to the injection for optimum positioning and technique.

2. Only one injection of the opaque medium is usually necessary, thus reducing the possible hazard to the patient. If a further introduction of opaque medium is necessary for special situations, such as delineation of an aneurysm, then only a second injection is required rather than three, four or more, as occurs with single plane examinations.

3. Positioning of the patient on a special mobile table is simple and considerably

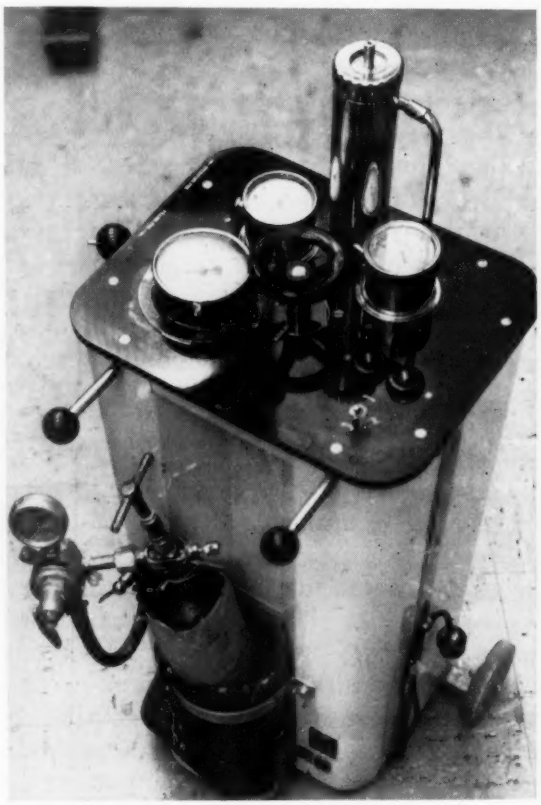

FIG. 3. The matching compressed air steel syringe (Gidlund) is shown.

easier than with the other serialographic units with which we are familiar.

4. A large film area (14×14 inches) in each plane permits examination of the entire skull including the face and also the major portion of the neck; the carotid bifurcation is thus always seen and technical failures such as extravasation, intimal injections and temporary spasm are easily determined. In addition, differential filling of the internal and external carotid artery circulation is readily demonstrated.

5. Great flexibility in sequential timing is available.

6. A three-dimensional reconstruction of the vascular system, not available with single plane studies, is readily apparent on the visualization of simultaneously exposed biplane roentgenograms.

7. There is magnification of small vessels which hitherto on smaller film areas were difficult to define. There is no accompanying significant loss in clarity.

8. The examination time is materially shortened.

9. Cut film (as used in our unit) is preferable to roll film (as used in other units) because of the ease of processing (Eastman-Kodak X-Omat), preparation, viewing and storage.

10. The adaptation of the matching high-pressure automatic injector will permit a marked reduction in radiation to the operator.

Figure 4, *A* and *B* is a representative frontal and lateral view of a normal cerebral angiogram performed with our biplane apparatus. Figures 5 through 8 are characteristic roentgenograms in a series of different disease states.

DISADVANTAGES

The increase in object-film distance with the use of the biplane Schönander unit in cerebral angiography results in some diminution in sharpness of the vessel walls. Thus, although vessel detail is sharpest in serialography when the head is in direct contact with the filming surface, there are enough advantages in our biplane unit to make this technique preferable. There is no question but that a single Bucky roentgenogram of the skull, exposed carefully with proper coning and less kilovoltage, will pro-

Fig. 4. (*A*) Frontal and (*B*) lateral 14×14 inch simultaneously exposed biplane roentgenograms in the arterial phase of a normal cerebral angiogram.

FIG. 5. (*A*) Frontal and (*B*) lateral simultaneously exposed biplane roentgenograms in a case showing a subdural hematoma.

FIG. 6. (*A* and *B*) A large aneurysm is demonstrated in the left middle cerebral artery at the junction of its first and second portions in these frontal and lateral roentgenograms with biplane technique.

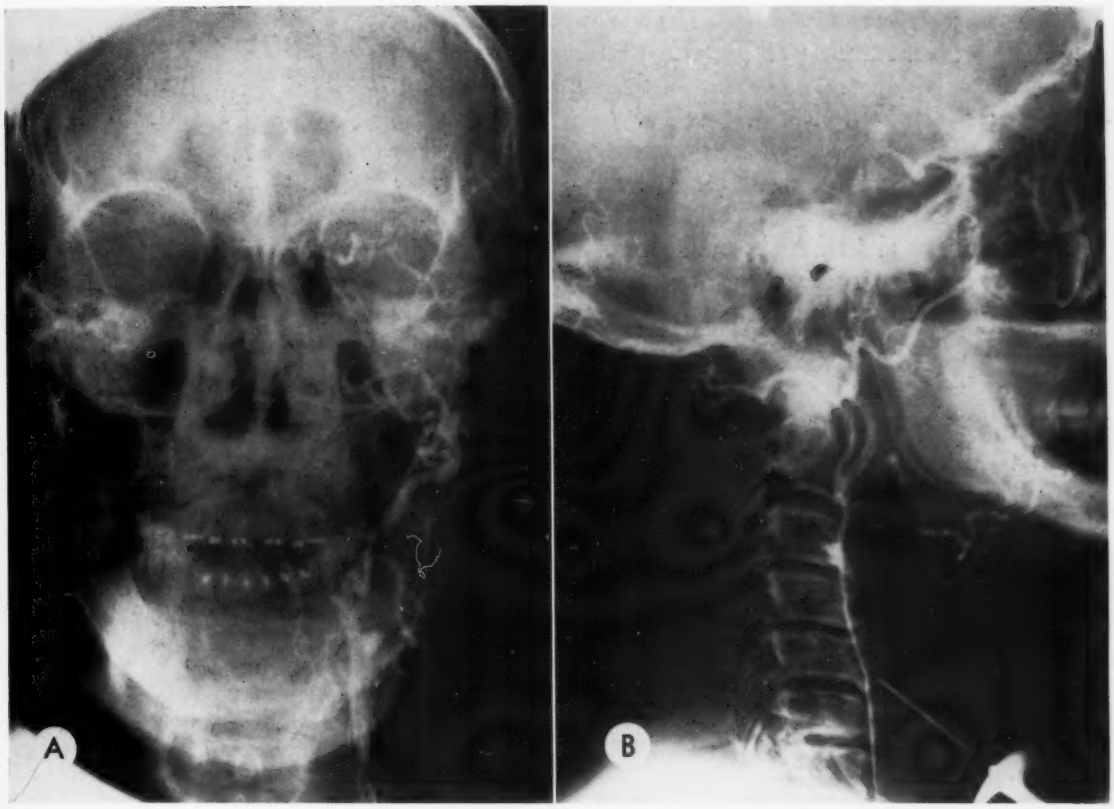

Fig. 7. (*A* and *B*) Occlusion of the left internal carotid artery with opacification of the external carotid circulation. There is retrograde opacification of the carotid siphon via the ophthalmic artery. Note the excellent delineation of the vessels in the neck, which is one of the major advantages of this method of examination.

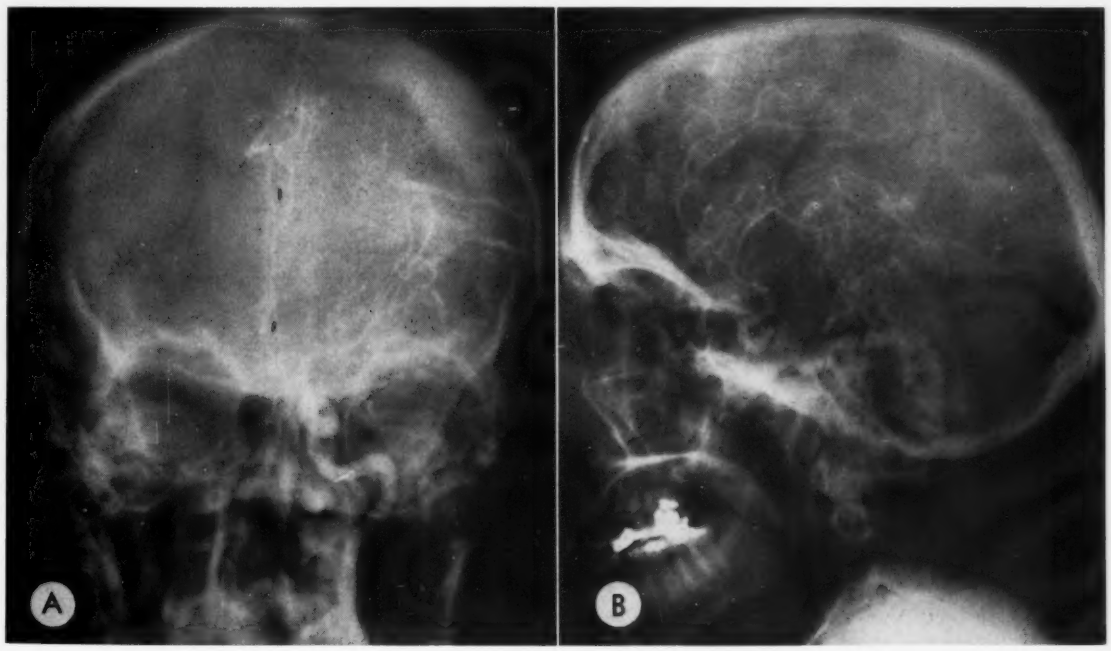

Fig. 8. (*A* and *B*) A glioblastoma multiforme in the left temporal lobe manifested by the characteristic displacement of the middle cerebral artery and a large tumor stain in this simultaneous biplane examination.

duce a more desirable roentgenogram than any seen in our method of biplane angiography or possibly even in any other form of rapid serialography. Again, there are obvious advantages in stereoscopy which are, as yet, not available to us with this unit.

In spite of the above-mentioned disadvantages, we feel that the advantages as noted are, in total, of such importance that our present method of cerebral angiography is desirable and even preferable to any other method currently available.

SUMMARY AND CONCLUSIONS

1. The utilization of the biplane Schönander unit in cerebral angiography is described and the technical details are included.

2. The advantages and disadvantages of this method of examination are discussed and it is felt that this technique is capable of producing optimal results.

3. Illustrative material representative of this technique is presented.

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EXPERIENCE WITH TWO NEW MEDIA IN ANGIOCARDIOGRAPHY

A PRELIMINARY REPORT

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ANGIOCARDIOGRAPHY is accepted as an important aid in the diagnosis and study of conditions affecting the heart, the lungs and the contiguous great vessels. Advances which have been made in the clinical application of angiocardiography have depended in part upon improvements in procedural technique, but even more, perhaps, upon the development and introduction of new and improved agents which have afforded better contrast with greater safety to the patient. To those familiar with the procedure, it is evident that the contrast material is responsible for the majority of the reactions which have occurred.[4,5,6]

HISTORY

Historically, the pioneer studies of Forssmann[7] were followed by the reports of others concerning the injection of radiopaque substances into the venous system to study the heart and the pulmonary circulation.[1,3,8] In 1937, Castellanos and his coworkers[1,2] devised a technique to which they gave the name "angiocardiography," in which the intravenous administration of uroselectan B was followed by a single exposure of the chest to roentgen rays during the termination of the injection. By this method it was possible to demonstrate the mediastinal veins, the chambers of the right heart, and the pulmonary arteries in children.[1] The following year, Robb and Steinberg[9] reported a method of cardiovascular visualization which consisted of the rapid injection of 70 per cent aqueous iodopyracet (diodrast) into a peripheral vein, with the patient in sitting position for roentgenography, and films exposed at the moment the contrast agent arrived in the pulmonary blood vessels or left heart. Films exposed three to five seconds after the beginning of injection usually demonstrated the right auricle, the right ventricle and the pulmonary arterial tree. The interval required for the medium to reach the left chambers was found to vary from six to sixteen seconds as determined by the cyanide circulation time. The fundamentals of the method of Robb and Steinberg are continued in present-day procedures though modern methods include devices for serial roentgen exposures of the chest which have lessened the need for precise timing of exposures. In the years since Robb and Steinberg reported their technique, a number of new contrast agents have been developed and introduced and the search continues for the ideal medium for use in angiocardiography.

MATERIAL

The present report concerns preliminary results with two new contrast agents specially designed for use in angiocardiography. The first of these to be offered for clinical trials, cardiografin 85 per cent, has been employed routinely in angiocardiographic studies conducted at this hospital since April 30, 1957, as well as in several cases in which unusual conditions were present. The second, cardiografin 90.5 per cent, which has a higher iodine content, has been used in angiocardiographic studies since January 15, 1958, in cases of cardiac lesions in the presence of an abnormal shunt or of an abnormal dilatation of the pulmonary artery where a great deal of dilution of the medium takes place. Cardiografin 85 per cent has proved to be very valuable in the diagnosis of congenital cardiac lesions in children and cardiografin 90.5 per cent has generally

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provided diagnostic roentgenograms with excellent delineation in those cases in which it was employed. A brief description of these preliminary results follows.

METHOD OF STUDY

The Contrast Agents. Cardiografin 85 per cent is a clear, sterile, aqueous solution containing 85 per cent of the methylglucamine salt of 3,5-diacetylamino-2,4,6-triiodobenzoic acid (diatrizoate). The iodine content of the solution is 40 per cent, and its viscosity (Brookfield) is 35.5 centipoises at 25°C.

Cardiografin 90.5 per cent is an aqueous suspension containing 60 per cent of the methylglucamine salt and 30.5 per cent of the sodium salt of 3,5-diacetylamino-2,4,6-triiodobenzoic acid (diatrizoate). The iodine content of cardiografin 90.5 per cent is 46.5 per cent, and the viscosity is 44 centipoises at 25°C.

The Patients. Up to the time of this report, a total of 29 patients has been examined with the cardiografins, as summarized in Table 1. Eighteen were female and 11 male. The oldest patient examined was eighty-four years of age; the youngest, eleven months. Eleven of the patients were under sixteen years of age, and, of these, 5 were twenty-four months of age or younger. Cardiografin 85 per cent was employed in the examination of 18 of the 29 patients, and cardiografin 90.5 per cent was used in the remaining 11 cases. All of the examinations performed with cardiografin 90.5 per cent were angiocardiographic studies. Of the 18 examinations in which cardiografin 85 per cent was the medium used, 15 were angiocardiographic studies. The 3 remaining examinations were: an oral study of the esophagus where there was a question of esophageal perforation and a sterile aqueous medium was needed; a translumbar aortographic examination of a patient with a large abdominal aneurysm where sufficient contrast could not be obtained with intravenously administered cardiografin; and a study of a fistulous sinus tract in the neck of a patient where a sterile, water-soluble medium was required.

Procedure. Angiocardiography usually followed cardiac catheterization in the patients with congenital heart disease. The contrast medium was injected by hand through a Robb-Steinberg needle (No. 15 or No. 12). One cubic centimeter per kilogram of body weight was the average dose for infants and young children. Fifty cubic centimeters of contrast material were the usual adult dose. Exposures were made with a Fairchild camera at a rate of 3 exposures

TABLE I

CONTRAST STUDIES WITH CARDIOGRAFINS: 35 INJECTIONS IN 29 PATIENTS

Contrast Agent Used	Type of Examination	No. of Patients	Sex		Age	Dose	Route of Administration	No. of Administrations
			M	F				
Cardiografin 85%	Angiocardiography	15	3	12	11 mo. to 84 yr.	15 cc. to 120 cc.	Intravenous	16
	Translumbar Aortography	1	1	0	79 yr.	20 cc.	Intra-arterial	1
	Opacification of Fistulous Tract in Neck	1	0	1	—	2 cc.	Injection into Sinus Tract	2 (1 cc. each)
	Esophagram	1	1	0	63 yr.	50 cc.	Oral	2 (50 cc. each)
Cardiografin 90.5%	Angiocardiography	11	6	5	11 mo. to 79 yr.	15 cc. to 100 cc.	Intravenous	14

every two seconds, usually with the patient lying on the table.

Anesthesia. For infants and children, pentobarbital sodium (nembutal) 3–4 mg./lb., together with morphine sulfate, 1 mg./10 lb., and scopolamine, 1/300 to 1/350 gr., were administered intramuscularly a half hour before catheterization and therefore usually 1.5 hours before angiocardiography. No consistent medication was used in adults. Barbiturate or meperidine was occasionally administered to anxious patients.

RESULTS

Diagnostic roentgenograms were obtained in most instances with both cardiografin 85 per cent and cardiografin 90.5 per cent. Cardiografin 85 per cent proved to have great diagnostic value in congenital cardiac lesions in children. In the study of pulmonary lesions in adults, this medium provided satisfactory to excellent opacification of the entire cardiovascular system including the left ventricle and aorta. Satisfactory contrast was also obtained in the 3 studies in which cardiografin 85 per cent was administered orally, intra-arterially, and directly into a sinus tract in the neck. In the case where a perforation of the esophagus was suspected, there was an excellent outline of the esophagus which established the absence of a perforation, while in the sinus tract study there was very adequate opacification of a branchial fistula in the neck. Likewise, the aorta and its branches were demonstrated satisfactorily in translumbar aortography.

Cardiografin 90.5 per cent proved to be valuable in children with congenital cardiac lesions in the presence of a left to right shunt as well as in adults with an abnormal shunt or an abnormal dilatation of the pulmonary artery, where a great deal of dilution of the contrast material takes place. In these cases, cardiografin 90.5 per cent because of its higher iodine concentration provided diagnostic contrast. Dramatic diagnostic results were seen in one case examined with cardiografin 90.5 per cent where an entire hypernephroma was opacified through the use of a nephrogram (Fig. 1).

Fig. 1. A hypernephroma is outlined after the injection of cardiografin 90.5 per cent.

Complications. There were no serious side reactions and no serious sequelae were seen following the administration of either cardiografin. A generalized urticaria with angioneurotic edema developed in one patient examined with cardiografin 90.5 per cent, which required the administration of adrenalin. This patient gave a history of rose fever but of no other allergies and did not react to the test dose of 1 cc. given prior to examination. Temporary cardiac standstill occurred in one infant with mongolism and pentalogy of Fallot after 25 cc. of cardiografin 85 per cent had been injected, but this standstill may have resulted from additional intravenous barbiturate which was required. Otherwise, side reactions were minimal. Flushing and a sensation of warmth was experienced by half of the patients examined with cardiografin 85 per cent but by only one to whom cardiografin 90.5 per cent was administered. (All of the patients except 2 who were given cardiografin 90.5 per cent were children and were

TABLE II

SIDE EFFECTS IN 29 PATIENTS EXAMINED WITH CARDIOGRAFIN 85% OR CARDIOGRAFIN 90.5%

Side Effect	Contrast Agent Employed		Remarks
	Cardiografin 85%	Cardiografin 90.5%	
Flushing	7	1	
Sensation of Warmth	5	1	
Urticaria	0	1	A generalized urticaria with angioneurotic edema developed which responded to adrenalin within 5 minutes
Cardiac Standstill	1	0	Standstill may have resulted in part at least from the anesthetic administered
Nausea	2	0	
Retching	1	0	
Coughing	1	0	
Total	17*	3	

* More than one side effect was experienced by several patients.

asleep at the time of the examination.) A fleeting sensation of nausea was experienced by 2 patients examined with cardiografin 85 per cent, and another had a coughing episode. The incidence of side effects which developed in the whole series appears in Table II.

DISCUSSION

The findings of this study indicate that both cardiografin 85 per cent and cardiografin 90.5 per cent fill a distinct need in the armamentarium of the cardiologist for the diagnosis of cardiac lesions and related conditions in children and in adults as well. Because of the physical properties of these media, certain distinct advantages attend the use of each. The lower viscosity of cardiografin 85 per cent permits its injection through needles of smaller bores than would be possible with cardiografin 90.5 per cent and other more viscous media. The use of smaller needles is a distinct advantage in children with small veins. The fact that the contained salt of cardiografin 85 per cent is highly soluble in water and does not crystallize out of solution at room temperature, as with other media, is another advantage, particularly if there is a delay from the time the solution has been warmed up to the time of injection. Also, cardiografin 85 per cent does not "freeze" the syringes, a property which makes it more desirable in selected cases, particularly in angiocardiography in children where rapid injection is essential for a smooth operation. On the other hand, because of the higher iodine content of cardiografin 90.5 per cent, its use is advantageous in situations where a great dilution of the contrast material takes place, as in large left to right shunts, and where a thick chest wall is a factor. Cardiografin 90.5 per cent, however, does not possess the advantage of high solubility of the contained salt.

The paucity of side reactions seen in this series is remarkable. This low incidence of reaction is one of the most advantageous properties of the cardiografins. The quality of contrast provided by these media combined with their comparative freedom from toxic manifestations makes them highly satisfactory contrast agents for use in angiocardiography.

SUMMARY

Two new, water-soluble contrast agents, cardiografin 85 per cent and cardiografin 90.5 per cent have been employed in angiocardiographic and other roentgenographic diagnostic studies in a total of 29 patients. With few exceptions, diagnostic roentgenograms were obtained with each contrast medium. Cardiografin 85 per cent proved to have great diagnostic value in congenital cardiac lesions in children. In the study of pulmonary lesions in adults, for the most part there was satisfactory to excellent opacification of the entire cardiovascular system, including the left ventricle and aorta. In the presence of a large left to right shunt with dilution of the contrast medium in an enlarged right heart and in thick-chested adults, cardiografin 90.5 per cent with its higher iodine content provided better and more diagnostic contrast than did cardiografin 85 per cent. However, the lower viscosity of cardiografin 85 per cent permitted its injection through needles with

smaller bores, a distinct advantage in children; and the high solubility at room temperature of the contained salt of cardiografin 85 per cent prevented its crystallizing out and the "freezing" of the syringes in which it was administered, a fact which contributed to smoother technique and more satisfactory results.

No serious side reactions were seen in the entire series. Only one case required the administration of adrenalin because of the development of a generalized urticaria. Transient cardiac standstill occurred in one mongoloid with pentalogy of Fallot but this effect may have resulted from respiratory depression due to supplementary intravenous barbiturate. Otherwise, the side reactions were minimal, consisting principally of flushing and a sensation of warmth.

The quality of contrast provided by the cardiografins and the paucity of unwanted side reactions attending their use indicate that they are highly satisfactory media for use in angiocardiographic and other intravenous roentgenographic procedures for the diagnosis and study of cardiac and related conditions in both children and adults.

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PELVIC ANGIOGRAPHY IN DISEASES OF THE PROSTATIC GLAND

By A. BOBBIO, E. BEZZI,* *and* L. ROSSI†

ANGIOGRAPHY, which for many years has been restricted to investigation of vascular disease, has been employed recently with increasing frequency for the study of other diseases as well as anatomic structures. It has been used in preoperative localization of parathyroid adenomata,[14] in the study of coronary circulation,[7] in gynecologic[7,13] and obstetric[6] conditions, in bladder diseases,[12] and in thyroid pathology.[2,3]

This increased use is the result of vast experience and intensive research work and is enhanced by the availability of better contrast media, improvement in the technique of arterial injections, and development of serial roentgenography. Angiography has now become a reliable diagnostic procedure not only for morphologic studies but also for the functional evaluation of vessels. To secure the maximum information one must have a good knowledge of the circulatory anatomy and physiology of the region concerned. Too, the technique of injection must be adapted for the particular area.

In general, two methods allow the outlining of a circulatory district: (1) the rapid injection of contrast medium into a large artery, the branches of which supply the entire region to be investigated; (2) the injection of contrast medium into a small artery which supplies a given anatomic structure by introducing a polyethylene tube into a larger artery up to the point where the small artery originates. Both of these methods have advantages and disadvantages. Some vessels are better explored by catheterization. Among these are the coronary arteries, the celiac trunk, etc. Other vessels are best studied by arterial puncture. This has the obvious advantage of being a simpler procedure. Rossi and coworkers[12] have stressed the importance of serial roentgenograms taken at precisely chosen intervals so that, by combining the morphologic and functional findings of a given structure, a correct differential diagnosis may be made.

Mindful of these possibilities, we have investigated the value of arteriography in the diagnosis of prostatic diseases.

THE TECHNIQUE OF PROSTATIC ANGIOGRAPHY

Of the different techniques of pelvic angiography, we chose retrograde iliacography by puncturing the femoral artery, instead of selective arteriography by catheterization or translumbar aortography. The retrograde puncture of the femoral artery allows good visualization of the pelvic vessels, is simple and comparatively innocuous.

In our series, arteriography was performed with the patients under general anesthesia. Although the procedure could be well tolerated by the unanesthetized patient, we believe that anesthesia minimizes the local vasomotor reactions caused by the arterial puncture and the injection of a strongly concentrated contrast medium.

To obtain good concentration of the opaque material above the branching of the common iliac artery, the following rules should be adopted: (1) The puncture of the femoral artery should be performed by means of a large needle (No. 22–24 gauge); (2) the point of the needle should be introduced for one inch or so into the artery by directing it toward the heart; (3) an arterial tourniquet should be applied to the thigh of the concerned side while the contrast medium is injected; (4) the contrast medium should be injected rapidly (30 cc. within four to five seconds). We used urografin 70

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per cent as the contrast medium.

Prostatic arteriography should be performed on one side at a time by two separate procedures. Simultaneous bilateral injection hinders the retrograde flow of the medium toward the iliac branches. Both sides should always be explored since it is not possible to determine beforehand on which side the angiographic signs of disease will appear.

By means of an automatic serialograph, roentgenograms are taken at two and one-half, four and one-half, five and one-half, seven, nine and eleven second intervals while the contrast medium is being injected. This technique allows a complete visualization of the various phases of pelvic angiography.

NORMAL ANGIOGRAPHY OF THE PROSTATIC CIRCULATION

The prostatic gland is supplied chiefly by branches of the middle hemorrhoidal, lower vesical and internal pudendal arteries. These vessels are clearly outlined by the contrast medium and their course can be traced from their origin down to the finest ramifications.

The lower vesical artery originates from the internal iliac artery. It follows a vertical caudad course to the lateral border of the pelvis, where it turns horizontally toward the midline to reach the symphysis pubis; here, its level is either at the body of the os pubis or immediately above it (Fig. 1, *A* and *B*).

The middle hemorrhoidal artery origi-

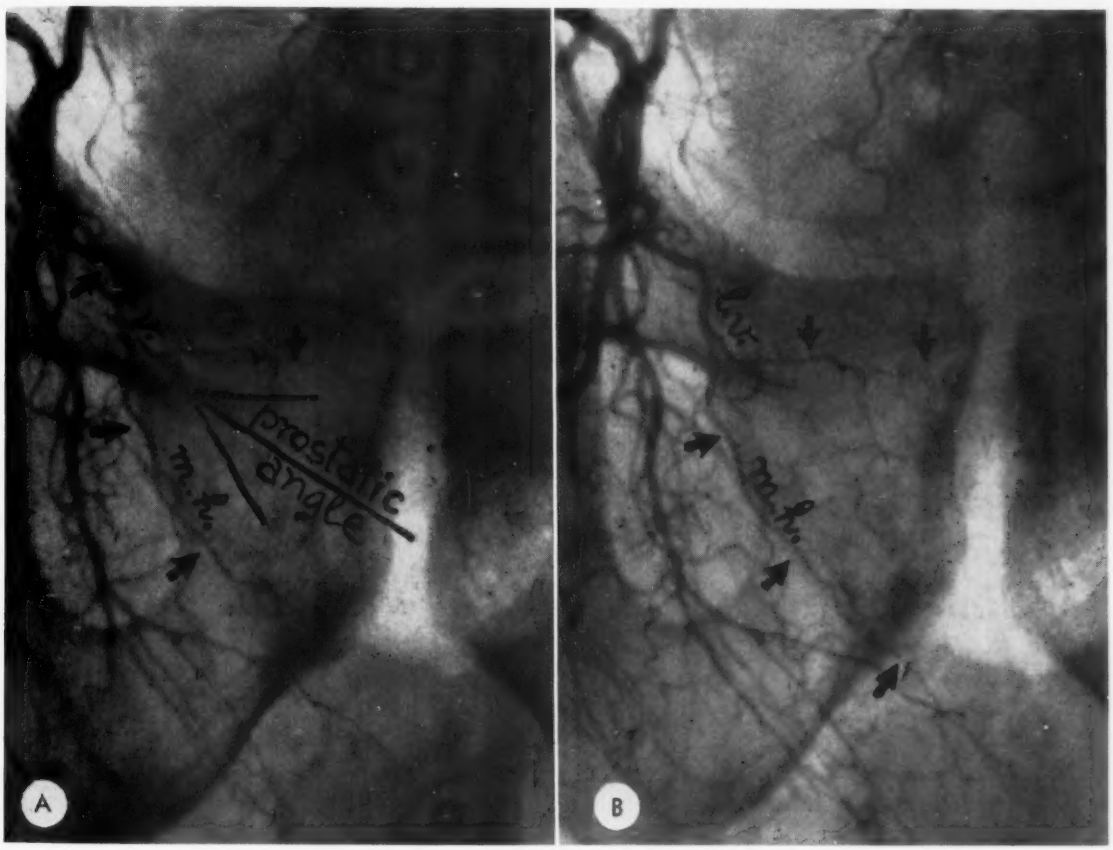

Fig. 1. *Normal Pattern.* (*A*) Early and (*B*) late arterial phases, two and one-half and five and one-half seconds, respectively, after injection of the contrast medium: l.v., lower vesical artery (from internal iliac artery); m.h., middle hemorrhoidal artery (from lower vesical artery). These vessels form an angle, called the "prostatic angle," at the site of the prostatic gland. This site is coursed by a number of fine vessels from both the lower vesical and the middle hemorrhoidal arteries, which are poorly outlined by the contrast material.

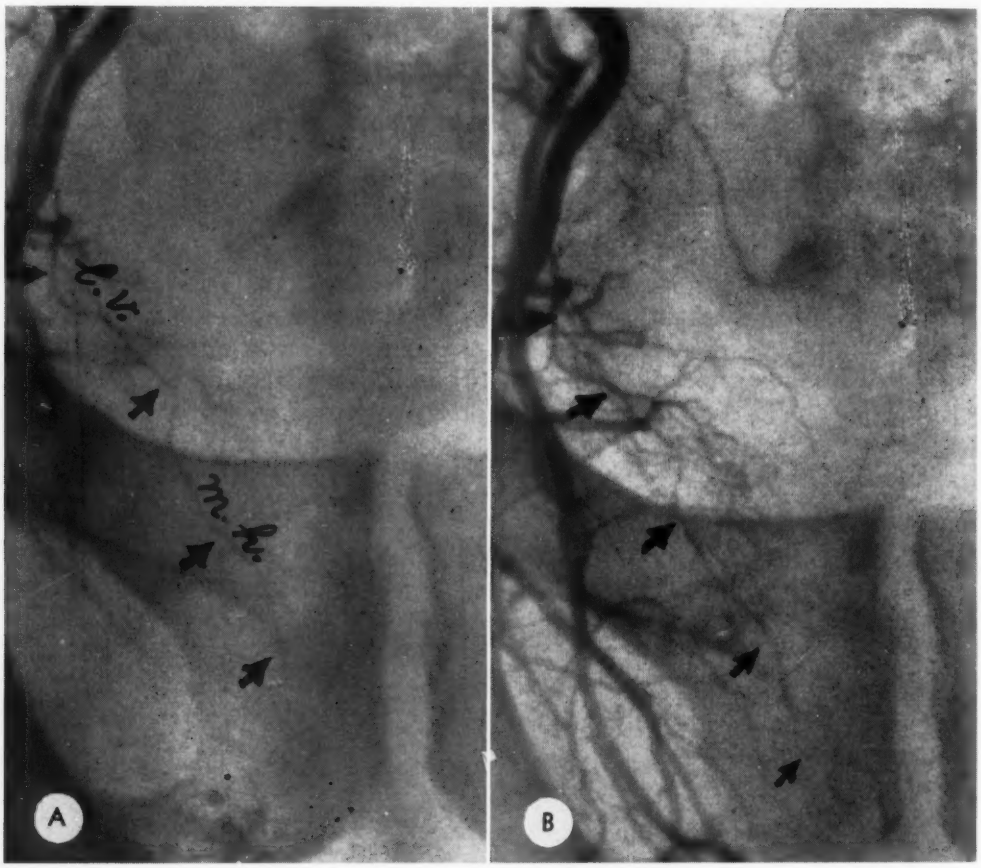

FIG. 2. *Chronic Prostatitis.* (*A*) Early and (*B*) late arterial phases. The arteries are finer than normal and poorly visualized.

nates from the internal iliac artery (in 11 out of our 19 cases), the internal pudendal artery (2 cases), or from the lower vesical artery (2 cases). In 4 of our cases it was not outlined. It runs downward and toward the medial border of the pelvis, crosses vertically the horizontal branch of the os pubis at its middle third, and then reaches the lower border of this bone structure.

From both the lower vesical and the middle hemorrhoidal arteries arise several very fine vessels which supply the prostatic gland. Normally, these arterial branches are poorly outlined by the contrast medium because of their small size.

During the capillary phase of angiography (seven to thirteen seconds after injection), the normal prostate is not stained by the contrast medium as are the kidneys and spleen. The liver, adrenal glands, and uterus are similar to the prostatic gland in this respect.

The venous phase is never seen in the normal prostatic gland.

PROSTATIC ADENOMA

The angiographic signs of prostatic adenoma (Fig. 3, *A* and *B*, and 4) are as follows:

(a) The lower vesical artery, after descending along the medial border of the pelvis, is deviated from its normal horizontal course by an angulation which varies from a few degrees to 180 degrees. It then follows an upward oblique course toward the midline, forming curves of various sizes. This topographic pattern is due to the fact that the lower vesical artery is raised by the enlarged prostate. It corresponds to the cystographic finding of a rise of the lower

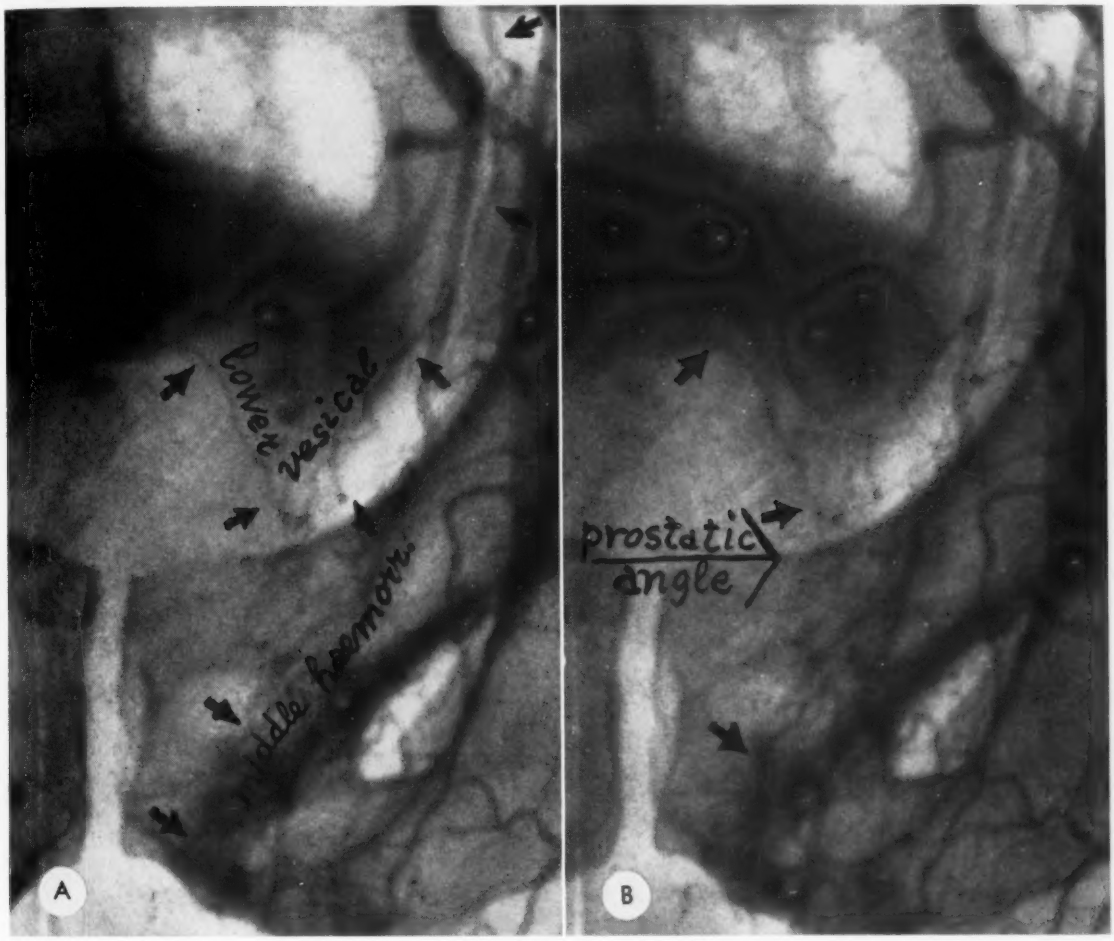

Fig. 3. *Prostatic Adenoma.* (*A*) Early and (*B*) late arterial phases. The lower vesical artery, after coursing downward along the border of the pelvis, is sharply displaced upward and laterally. The prostatic angle is wider than normal.

profile of the bladder in the presence of prostatic adenoma.

(b) The middle hemorrhoidal artery may also be displaced at the level of its retropubic portion. This finding, which is less frequent than the rise of the lower vesical artery, is due to enlargement of the prostatic gland.

(c) In some cases very fine arterial vessels are also outlined, originating from the lower vesical artery and spreading upward, fan-like, along the border of the prostatic growth. This is probably due to inflammatory hyperemia of the bladder, which is often assocated with prostatic dysuria.

The capillary and venous phases have not been seen in cases of prostatic adenoma.

MALIGNANT TUMORS OF THE PROSTATE

The angiographic signs of a malignant tumor of the prostate (Fig. 5; 6, *A–C;* 7, *A–D;* and 8, *A* and *B*) are as follows:

(a) The prostatic arteries (lower vesical, middle hemorrhoidal and internal pudendal arteries) are large and tortuous, giving origin to a number of fine, irregular, small vessels which spread medially, fan-like, into the neoplastic area. In cases where the prostatic gland is larger than normal, these arteries present the same topographic displacements that are seen in the presence of prostatic adenoma.

(b) The anastomosing pattern of the vessels has no regular distribution. It forms a thick vascular network, the caliber of the

FIG. 4. *Prostatic Adenoma.* Late arterial phase. From the lower vesical and the middle hemorrhoidal arteries originate a number of fine vessels (prostatic arteries) which encircle, fan-like, the convex surface of the prostatic growth. The prostatic angle is widened.

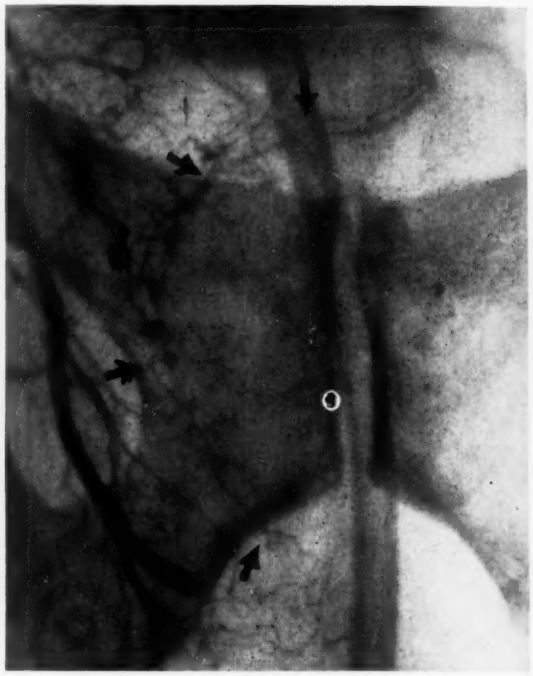

FIG. 5. *Carcinoma of the Left Lobe of the Prostate.* Right iliacography. Late arterial phase. The angiographic picture is that of a prostatic adenoma. The prostatic angle is widened. No pathologic vascularization of the right prostatic lobe is seen. Postoperative histologic examination confirmed that this side of the prostate gland showed no sign of malignancy. (See also Fig. 8.)

vessels and the anastomosing branches being completely irregular.

(c) In the arteriolar phase of angiography, intratumoral pooling can be observed.

(d) Between seven and twelve seconds, a direct staining of the neoplastic tissue is noted. This stain, however, is not homogeneous and occurs in patches of varying opacity.

(e) The venous drainage of the opaque blood from the tumor occurs earlier than it does in normal cases or in adenoma.

A venous phase can be seen clearly between seven and twelve seconds. The intratumoral veins, as well as the vesicoprostatic and pudendal plexuses, may be outlined by the contrast medium in this phase of angiography (Fig. 6*C*; 7*D*; and 8, *A* and *B*).

CHRONIC PROSTATITIS

The angiographic appearance of chronic prostatitis differs very little from that of the normal prostate. In patients of advanced age who have developed sclerotic changes, the caliber of the lower vesical and middle hemorrhoidal arteries may be somewhat diminished, and the angle between these arteries may be narrowed. Capillary or venous phases have never been demonstrated by serialography (Fig. 2, *A* and *B*).

CONCLUSIONS

The findings in prostatic angiography are not as outstanding as the findings in angiography of other organs. In spite of this, the method has allowed us to elicit some signs of clinical value. In the presence of prostatic adenoma, we have observed a widening of the angle between the lower vesical and middle hemorrhoidal arteries which encircle the lateral border of the prostate. The capillary and venous phases, as in the normal prostate, are not visualized.

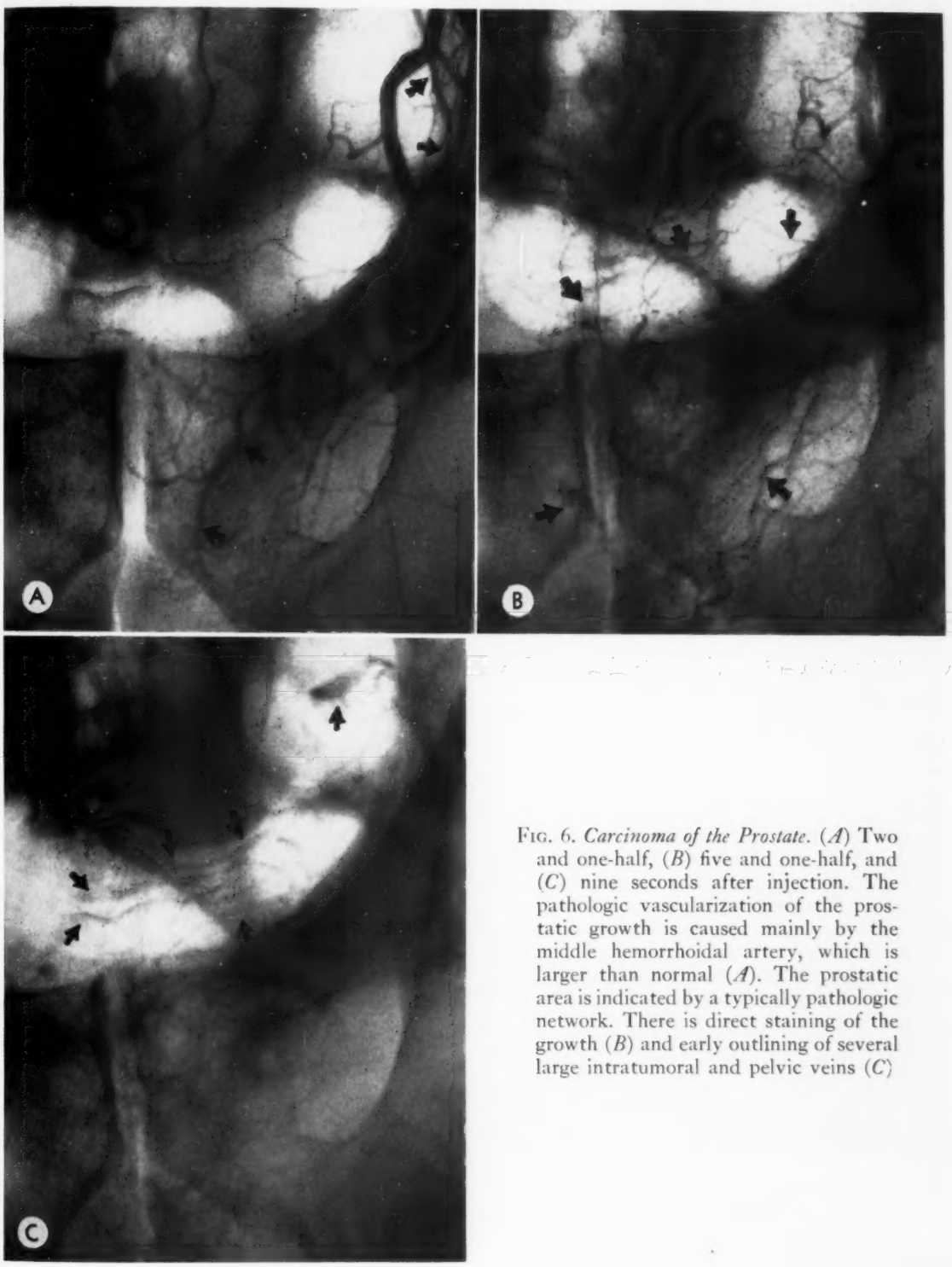

Fig. 6. *Carcinoma of the Prostate.* (*A*) Two and one-half, (*B*) five and one-half, and (*C*) nine seconds after injection. The pathologic vascularization of the prostatic growth is caused mainly by the middle hemorrhoidal artery, which is larger than normal (*A*). The prostatic area is indicated by a typically pathologic network. There is direct staining of the growth (*B*) and early outlining of several large intratumoral and pelvic veins (*C*)

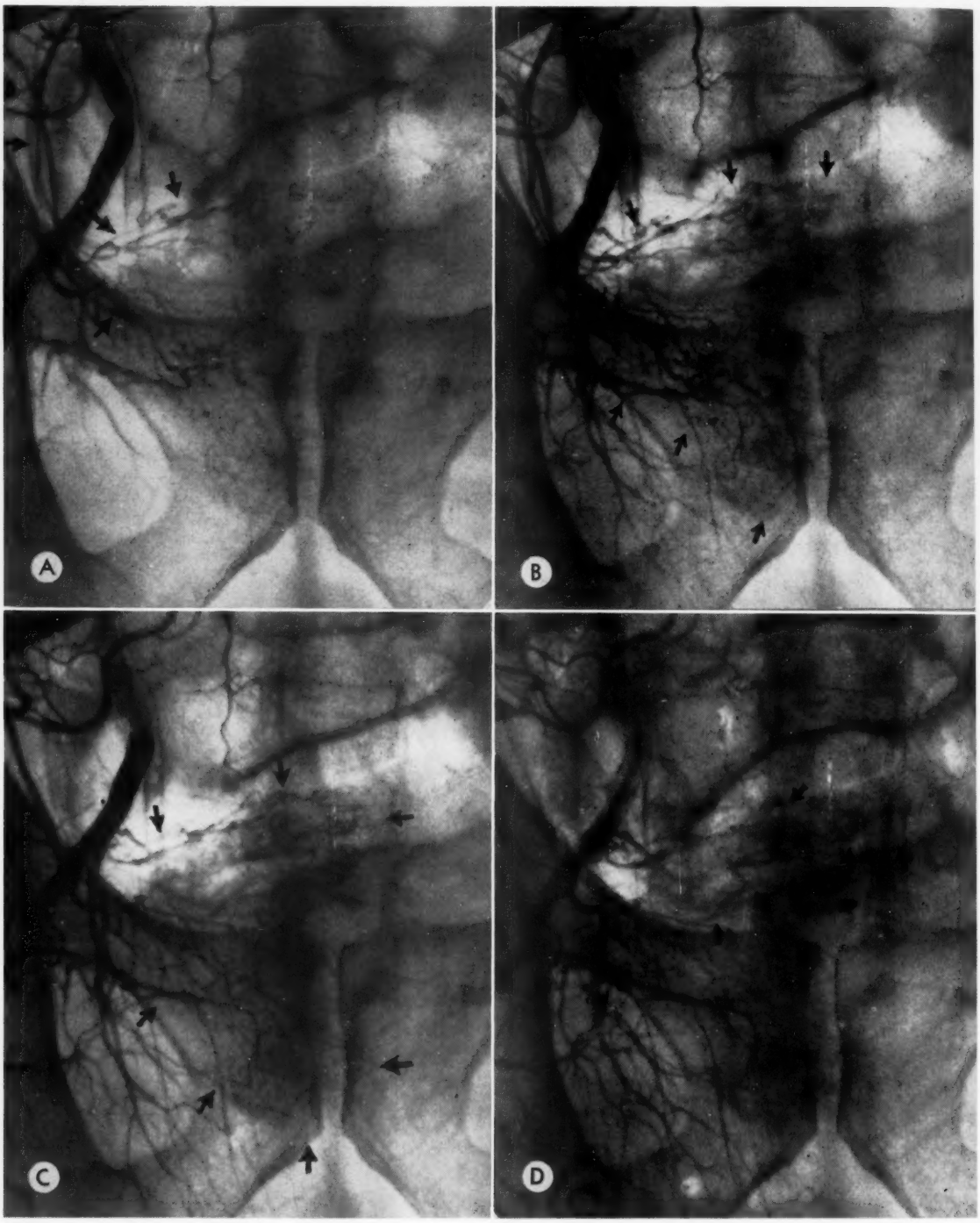

FIG. 7. *Carcinoma of the Prostate.* (*A*) Two and one-half, (*B*) five and one-half, (*C*) seven, and (*D*) nine seconds after injection. The lower vesical artery branches off into three rami, which enter the prostatic growth, anastomosing irregularly. Also the prostatic and the middle hemorrhoidal arteries, larger than normal, take part in the irregular network which covers the whole prostate and crosses the midline. There is direct outlining of the growth in the late arterial phase (*C*) and visualization of the veins after only nine seconds following injection of the contrast material (*D*).

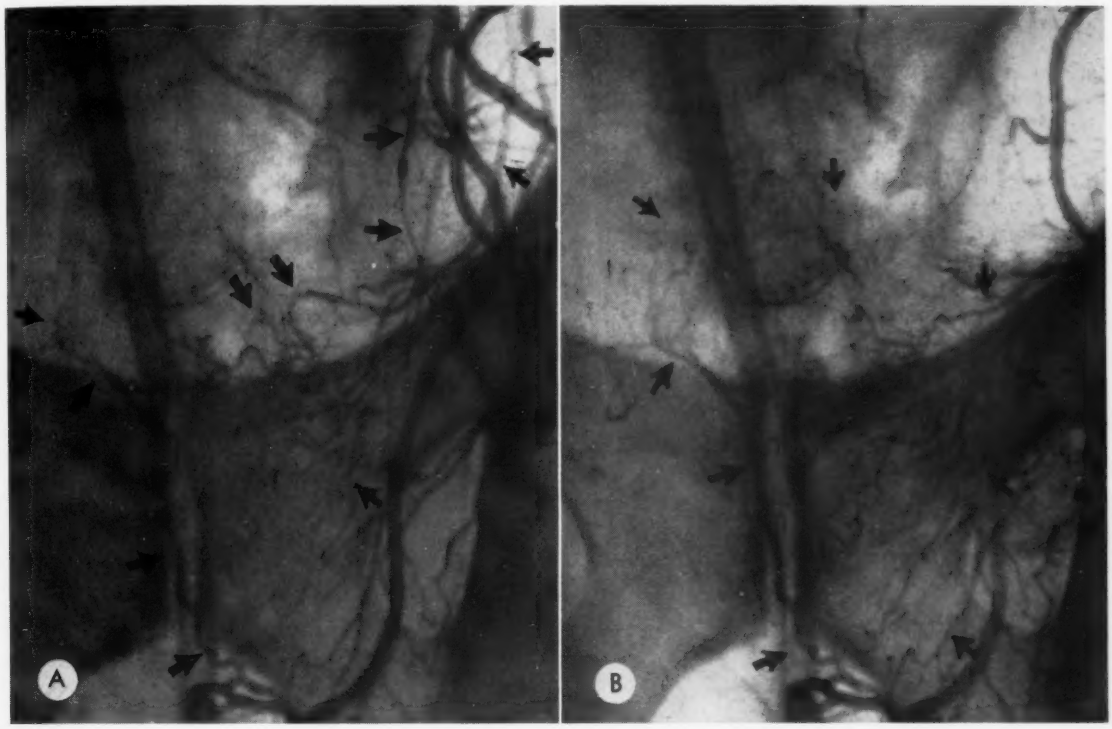

Fig. 8. *Carcinoma of the Left Lobe of the Prostate.* Left iliacography. (*A*) Early and (*B*) late arterial phases. Four arteries (double lower vesical, middle hemorrhoidal and prostatic arteries) which course into the growth and anastomose with each other following an irregular pattern are outlined. A big branch of the internal pudendal artery ascends to the prostatic area past the midline. The entire growth is also outlined by the contrast material (*B*).

Chronic prostatitis causes no particular changes in the morphology or functional aspects of the prostatic circulation except for some decrease of the blood supply when severe sclerosis of the gland has taken place.

In the presence of a malignant growth of the prostate, the angiographic pattern resembles that observed in malignant tumors of other organs, producing a rich, irregular blood supply, pooling of blood, staining of the parenchyma, and early venous drainage.

The results indicate that pelvic angiography, when correctly performed, may give useful information in the differential diagnosis of prostatic diseases.

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THE CAVUM VELI INTERPOSITI AND ITS DIFFERENTIATION FROM CAVUM VERGAE*

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THE purpose of this paper is to present the pneumoencephalographic picture of the so-called cavum veli interpositi, or interventricular cistern; to differentiate it from other structures of similar appearance; and to discuss its clinical significance.

A brief description of the embryologic development of the cavum and related structures is herewith given.[1,7,9,10,11,16] The tela choroidea of the third ventricle originates from the roof plate of the diencephalic region at about the third fetal month. A fold of pia mater protrudes into the primitive neural tube. With further development of the cerebral hemispheres and particularly the development of the corpus callosum, this fold, which originally is wide open, undergoes changes. The corpus callosum is formed in the rostral end of the midline of the telencephalon about the third month of pregnancy and grows posteriorly, reaching its definitive location at the end of the fifth fetal month. The fornices originate in the thin rostral wall of the third ventricle above the anterior commissure, migrating caudad, with, and beneath the corpus callosum. Concurrent with this development, the pia mater fold is pushed backward forming the final tela choroidea of the third ventricle including a horizontal sac or fissure under the fornix which opens behind and under the splenium of the corpus callosum where its pia mater is connected to the pia mater covering the median fissure of the cerebrum. This sac-like pia fold carries the name "transverse or choroidal fissure." The final tela choroidea assumes a triangular shape, its apex reaching anteriorly to the interventricular foramen (foramen of Monro); its base lying beneath the posterior end of the hippocampal commissure and the splenium of the corpus callosum; its lateral borders resting upon the medial wall of the thalamus. The tela choroidea of the third ventricle contains the two internal cerebral veins which unite posteriorly to form the great vein of Galen. In the majority of cases the choroid fissure closes, if not anatomically at least functionally. According to Robertson[12] this space normally obliterates, but the evidence points to the fact that while not demonstrable in a high percentage of pneumoencephalograms it is potentially distensible under certain conditions. Some of these conditions include hypoplasia of the corpus callosum, diseases of the corpus callosum, and tumors in this area producing distortion of the structures. When it persists, the choroid fissure is called the "cavum veli interpositi or cisterna interventricularis." This is a true cisternal structure communicating through the cisterna of the veins of Galen via the ambient and basal cisterns. The borders of the cistern of the vein of Galen are: anteriorly, the pineal gland; posteriorly, the cerebellum; rostrally, the splenium of the corpus callosum and the free edge of the tentorium cerebelli; and, inferiorly, the quadrigeminal plate. On both sides of the quadrigeminal plate this cistern enters the ambient cisterns.

Kruse and Schaetz[8] in 1935 for the first time were able to recognize the cavum veli interpositi (C.V.I.) on the pneumoencephalogram. These authors used the name—interventricular cistern or cavum. They described the case of a two and one-half year old girl with tuberculous meningitis who underwent several air studies. The interventricular cavum filled on three occasions; on one occasion only the basal cisterns and the C.V.I. were visible, the ventricular sys-

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tem failing to fill. Postmortem examination of this child revealed, besides other findings, a tuberculoma in the area of the quadrigeminal plate. The fornices and the psalterium were displaced upwards, thus opening the entrance into the interventricular cavum. This observation led to the conclusion that this structure belongs to the subarachnoidal and cisternal system, and that the air reaches it not through the ventricles, but the basal cisterns via the ambient cistern and the cistern of the vein of Galen. Robertson independently described this cavum in 1946[12] and again in 1957[13]. He found it in 8 cases of 500 pneumoencephalograms. Zellweger[20] in 1951 presented a series of 30 cases from 500 pneumoencephalograms performed in Children's Hospital in Zurich. Childe[3] in 1952 described the cavum as cisterna velum interpositum. He presented two photographs of brain sections of infants demonstrating the structure in addition to showing the pneumoencephalographic appearance of it. The present authors found 9 cases in the last three years in a study of 200 pneumoencephalograms.

ROENTGENOLOGIC DESCRIPTION OF CAVUM VELI INTERPOSITI

This structure is best identified in the lateral stereoscopic projections. When it is filled with air, the cavum veli interpositi lies above and posterior to the third ventricle. Its size, shape and degree of filling varies from case to case. When the cavum is large and well filled with air, its anterior extent lies behind the foramina of Monro, its posterior superior margin or border is indented by the splenium of the corpus callosum, its anterior border is separated from the third ventricle (if filled with air), and its inferior margin is indented by the great vein of Galen. Although the cavum veli interpositi fills via the ambient cisterns and the cisterna venae magnae cerebri, not always do we see these structures. When the cisterna venae magnae cerebri is filled with air, the C.V.I. is separated from it by a band of radiodensity produced by the great vein of Galen as it curves upwards and backwards around the splenium of the corpus callosum on its way to the straight sinus. These structures are well demonstrated in Figures 1 and 2. The quadrigeminal plate may be seen in the anterior aspect of a well filled cisterna venae magnae cerebri.

In the anteroposterior view the C.V.I. is an air filled structure of varying size and contour situated between parts 2 and 3 of the lateral ventricles beneath the fornices and above the third ventricle. The fornices, when visualized, are noted to hang down or droop from their usual horizontal position. The third ventricle, when it is well filled, may obscure the margins of the cavum. The opposite is also true. In our experience the lateral view gives the greatest aid in the detection of this cavum.

DIFFERENTIAL DIAGNOSIS

The cavum veli interpositi has to be distinguished primarily from cavum septi pellucidi and cavum Vergae. It is not difficult to distinguish a cavum septi pellucidi from a cavum veli interpositi, since in the lateral projection it lies in front of the foramen of Monro, whereas the cavum veli interpositi lies behind it. Since a cavum veli interpositi does not enlarge the septum pellucidum and, in fact, is divided in half by it as it is viewed in the anteroposterior projection, a transverse diameter of the septum pellucidum of more than 5 mm. indicates either the presence of a tumor or a cavum of the septum pellucidum, even if the latter does not communicate with the ventricular system and fails to fill with air.

It appears to be more difficult to distinguish the cavum veli interpositi from a cavum Vergae. Before going into any discussion of this differential diagnosis, it may be worthwhile to define what is meant by cavum Vergae, since this cavum has been defined differently by different authors.[8] For some, it is identical with the cavum septi pellucidi, particularly if the latter reaches further back than the anterior pillar of the fornix. Others identify only that part

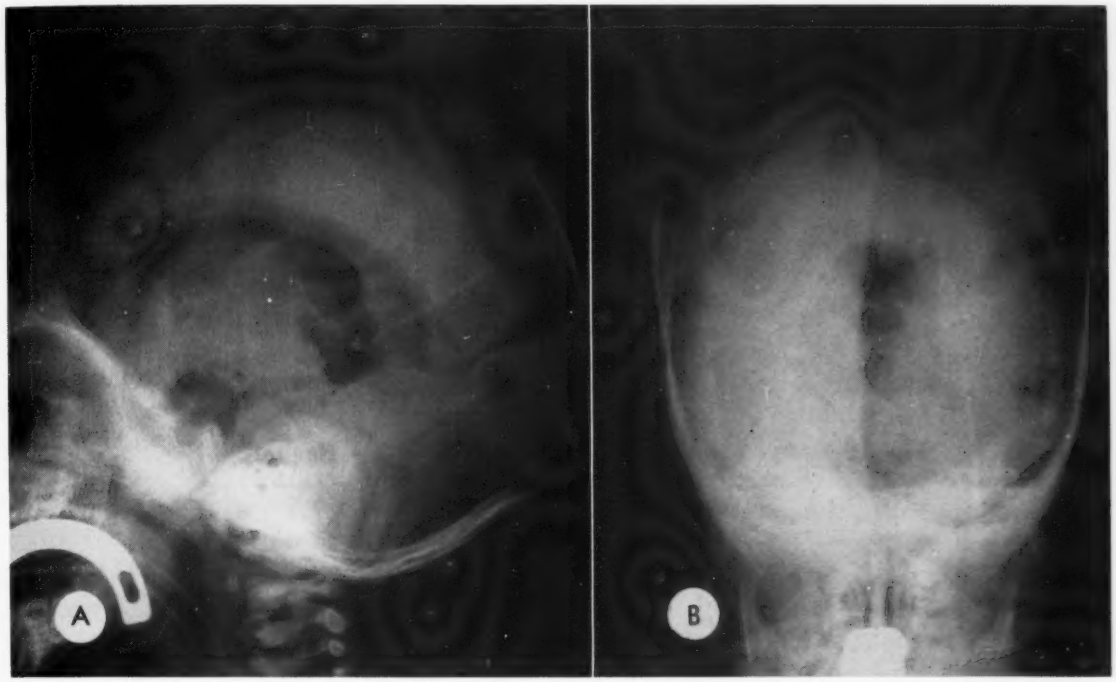

Fig. 1. Case I. (*A*) Pneumoencephalogram in the lateral projection shows a large oval-shaped cavum veli interpositi above and posterior to the third ventricle. It is separated from a triangular air filled cistern (cisterna venae magnae cerebri) by a band of radiodensity which is produced by the great vein of Galen. Note the quadrigeminal plate indenting the anterior surface of this cisterna. (*B*) In the anteroposterior projection the cavum lies between the lateral ventricles and above the third ventricle. In this case a diagnosis could not be made from this projection alone.

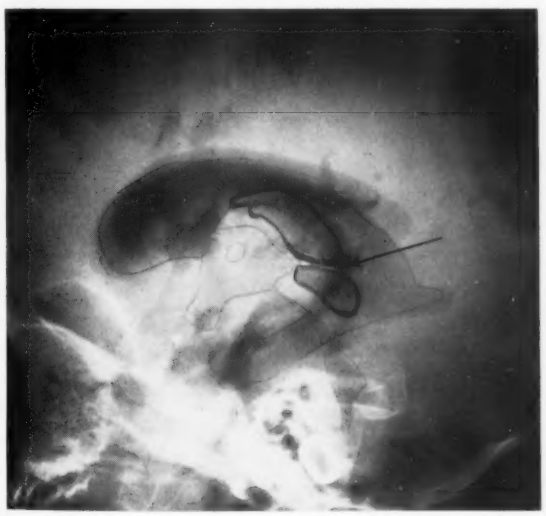

Fig. 2. Case II. Retouched lateral pneumoencephalogram demonstrates a large, somewhat oval-shaped cavum veli interpositi above and posterior to the third ventricle. The arrow points to the band of radiodensity between it and the cisterna venae magnae cerebri produced by the vein of Galen.

of the cavity lying behind the anterior limb of the fornix as a cavum Vergae. If we assume that the cavum Vergae includes only the cavum behind the anterior pillars of the fornix, the cavum would be related to the following structures: anteriorly, the aqueduct of the cavum septi pellucidi; superiorly, the lower surface of the corpus callosum; inferiorly, the upper surface of the hippocampal commissure; laterally, the posterior limbs of the fornices. This, we believe, is its correct description. Although the cavum Vergae is called the sixth ventricle, ventriculus triangularis or ventriculus fornicis, it is probably incorrect to call it a ventricle, because it does not originate from the primitive neural tube and is, therefore, not covered with an ependymal layer. It is a secondary cavity due to incomplete obliteration of the original cleavage between the fornices and the corpus callosum.

Schwidde[16] found a cavum septi pellucidi in 20.43 per cent and a cavum Vergae in 2.32 per cent of over 1,000 formalin fixed brains. In no instance was there a cavum Vergae without the simultaneous presence of a cavum septi pellucidi. He and others have noted, however, a cavum septi pellucidi without a cavum Vergae.[3] This is an important observation for the roentgenologic differentiation between a cavum Vergae and a cavum veli interpositi. This is true because in the absence of a cavum septi pellucidi, an air-filled space behind the foramen of Monro is more likely to be a cavum veli interpositi than a cavum Vergae. Since the hippocampal commissure is such a thin structure, the roentgenologic distinction of the cavum Vergae lying above and the cavum veli interpositi lying below it is impossible. It should be remembered that a cavum Vergae cannot reach further backward than the splenium of the corpus callosum, whereas the cavum veli interpositi occasionally reaches much further backward and merges with the cistern of the great vein of Galen, which in turn reaches down to the quadrigeminal plate. Several authors[5,6,19] have interpreted roentgenograms as showing cava Vergae which look very much like the roentgenograms in the present paper. We believe that Figure 221 in Davidoff and Epstein's[5] book represents a cavum veli interpositi rather than a cavum Vergae. The same holds true for Figures 5 and 6 in Zellweger and Hess'[19] article, and in several illustrations in Schönenberg's publication.[15] A postmortem examination in the case of Davidoff and Epstein[5] and in one case of Schönenberg's[15] report failed to reveal a cavum Vergae. Schönenberg in his article illustrates a transverse section through the region of the third ventricle to demonstrate the cavum which definitely is below the fornices and, thus, must be a cavum veli interpositi.

Other conditions which enter occasionally into differential diagnosis are a large suprapineal recess and the frontal sinus. The confusing shadow is first noted in the anteroposterior projection, and whereas it mimics the cavum veli interpositi, a careful study of the lateral projection indicates the true nature of the finding.

CLINICAL SIGNIFICANCE OF THE CAVUM VELI INTERPOSITI

The clinical significance of the cavum veli interpositi is not clear. In the following paragraphs an analysis of the clinical data of the cases reported in the literature and of the cases in this series will be given. We feel that 7 of the 8 cases reported by Schönenberg[15] and 2 cases of Echternacht and Campbell[6] can be considered as having a cavum veli interpositi.

Family History. A family history is available for 44 cases, including the present series. Consanguinity between the parents was found twice.[15,20] Family history of convulsions was present in 14 cases. The mother of Case IV is being treated for epilepsy. A brother of Case V has convulsions. Neurologic disorders, psychosis and mental retardation have been found in a number of the families. Friedreich's ataxia occurred in the brother of Case V. In one family a case of amyotonia congenita was reported and in several cases spina bifida with severe microor possibly anencephaly was observed. In 21 of 44 families the history was noncontributory.

Pregnancy and Delivery. Data are available in 38 cases for the course of pregnancy and delivery. Uterine hemorrhages during the first trimester were recorded twice. Signs of toxemia occurred in 5 cases, among them our Case III. One case of scarlet fever, and one case of pyelitis in the first two months of pregnancy were reported.[20] No history of abnormal delivery was found.

Birth Weight. Of the 38 cases with known birth weights, 7 cases had birth weights below 2,500 grams. Thus, prematurity is at least twice as high as in the average population.

Age and Sex Incidence. In our series 5 cases were below the age of six months, 2 cases between six and twelve months, one case 2 years and nine months of age and one adult forty-five years old. This observation

coincides with the statement of Robertson that cavum veli interpositi can be found in all age groups. Of a total of 51 cases taken from the literature and from this series, 21 were from birth to six months, 17 from six to twelve months, 8 from one to two years, 4 from two to five years, and one adult. The cavum veli interpositi was found more frequently in males than in females, yet the figures are too small to permit definite conclusions as to the sex incidence.

Prognosis. In the 39 cases where a follow-up is known, 13 died in infancy. The causes of death were: cardiac arrest during craniotomy, cerebral phlebothrombosis, tuberculous meningitis, bronchiolitis and diffuse sclerosis. Others died of convulsions and hyperpyrexia. Zellweger[20] in 1951 believed that a cavum veli interpositi implied a serious prognosis. The recent experiences of Robertson,[13] Schönenberg,[15] and ourselves would indicate that it does not mean a serious prognosis, since it is found occasionally in patients with normal intelligence and without any serious brain defects.

REPORT OF CASES

Case 1. J.H. Hospital No. 55-5171. This child, product of a normal pregnancy and delivery, started to convulse at the age of three days. The convulsions began on the right side and became generalized subsequently. The patient was admitted to the State University of Iowa Hospital at the age of six weeks, again at two and one half and six months. There were no abnormal neurologic findings. The electroencephalogram showed focal spikes in the left parietal and central area. The pneumoencephalogram in the lateral and anteroposterior views showed a normal ventricular system without displacement (Fig. 1, *A* and *B*). Above and posterior to the third ventricle was a cavum veli interpositi. Its posterior border was indented by the splenium of the corpus callosum. It was separated from a triangular-shaped cavity (cistern of the vein of Galen) by a band of radiodensity which we felt was produced by the vein of Galen as it passed upwards and backwards over the splenium of the corpus callosum. If only the anterposterior projection were available, one could not differentiate between a cavum Vergae and a cavum veli interpositi. However, study of the stereoscopic lateral projections identifies the structures accurately.

In spite of various anticonvulsive treatments, the convulsions did not subside. A craniotomy was performed at the age of six months, but the child died of cardiac arrest during the operation. Autopsy did not reveal any pertinent findings. No special attention was given to the area of the velum interpositum. *Impression:* Intractable convulsions. Cavum veli interpositi.

Case 11. P.P. Hospital No. 57-12041. The patient was born prematurely in the eighth month of pregnancy having a birth weight of 2,160 gm., and was admitted to the hospital at the age of seven weeks for minor convulsions. Physical examination at this time revealed a body weight of 3,600 gm., height of 52 cm., and a head circumference of 37 cm. Macroglossia was present. There was considerable muscular hypertrophy and muscular hypertonia with exaggerated deep tendon reflexes. Protein-bound iodine was normal. The electroencephalogram and muscle biopsy were normal. Serum calcium varied from 8 to 10.2 mg. per cent, without sign of tetany. Inorganic serum phosphates were between 6.6 and 7.4 mg. per cent. Pneumoencephalography in the lateral view demonstrated a large cavum veli interpositi behind and above the third ventricle (Fig. 2). In the anteroposterior view (Fig. 3) the cavum was well seen between the lateral ventricles superiorly. The septum pellucidum was noted to apparently divide the cavum, but, of course, did not have any contact with it. The fornix on the left was more vertical than usual. The ventricular system showed moderate dilatation.

At the age of six months there was still considerable muscular hypertrophy, exaggerated deep tendon reflexes and a positive Babinski sign. Serum calcium was 8.8 mg. per cent, inorganic phosphates 7.4 mg. per cent. At eleven months there was normal development, muscle hypertrophy was less pronounced, and no muscular hypertonia was present. Deep tendon reflexes were brisk, but not exaggerated. Rossolimo and Babinski signs were positive and negative, respectively. The patient was able to walk. *Diagnosis:* Congenital muscular hypertrophy. Cavum veli interpositi.

Case 111. D.L. Hospital No. 57-15317. The mother of this patient had hypertension and

Fig. 3. Case II. Pneumoencephalogram in the anteroposterior view shows the cavum between the lateral ventricles as a somewhat fan-shaped structure. The fornices are more vertical than normal. In addition, there is mild dilatation of the ventricular system and widening of the sulci.

edema during pregnancy. The patient was born in the State University of Iowa Hospital. Anterior fontanelle measured 60×80 mm., head circumference was 35 cm. He was brought back to the hospital because of failure to gain weight. At the age of six weeks his weight was the same as his birth weight, 3.2 kg. Body length had increased from 49 cm. at birth to 52 cm. at six weeks. Clinical and laboratory studies revealed possible bilateral polycystic kidneys. At six and one-half months he had gained to 5 kg. The height was 57 cm., head circumference 41 cm., fontanelle still 50×70 mm. Total solutes of urine were 330 mosm./liter, blood urea nitrogen 20 mg. per cent, creatinine 1 mg. per cent, urine normal. The child was slightly retarded, was not yet able to sit. No abnormal neurologic findings were noted. Pneumoencephalogram in the lateral view (Fig. 4) clearly showed a cavum veli interpositi above and posterior to the third ventricle. It was separated from the cisterna venae magnae cerebri by a band of radiodensity produced by the vein of Galen. In addition, there was enlargement of the fourth ventricle and cisterna magna. In the anterioposterior projection (Fig. 5) a diagnosis could not be made, although it was suspected from the position of the fornices

Fig. 4. Case III. A cavum veli interpositi is well demonstrated above and posterior to the third ventricle. It is separated by the vein of Galen from the cisterna venae magnae cerebri. Note the enlargement of the fourth ventricle and cisterna venae magnae.

Fig. 5. Case III. Pneumoencephalogram in the anteroposterior projection does not in itself identify the cavum because of insufficient filling. The upper outline is the cavum, the lower is the cisterna venae magnae and the band of radiodensity is produced by the vein of Galen.

Fig. 6. Case III. Retouched lateral pneumoencephalogram of Figure 4.

which were more curved than normally. The degree of filling was not sufficient in this projection for accurate identification. Figure 6 is retouched to accentuate the structures for identification. *Impression:* Cavum veli interpositi, dilatation of the fourth ventricle and cisterna magna, the latter indicating hypoplasia of the cerebellum; questionable kidney malformation; failure to gain weight and height.

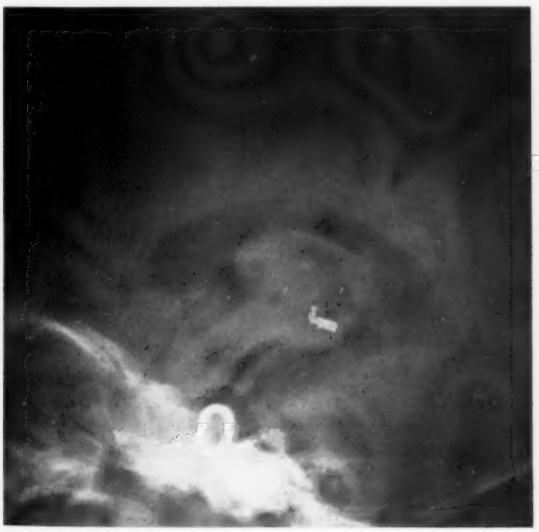

Fig. 7. Case IV. Pneumoencephalogram in the lateral projection showing a cavum veli interpositi above and posterior to the third ventricle. A cisterna venae magnae cerebri is also seen. It is separated by the vein of Galen from the cavum.

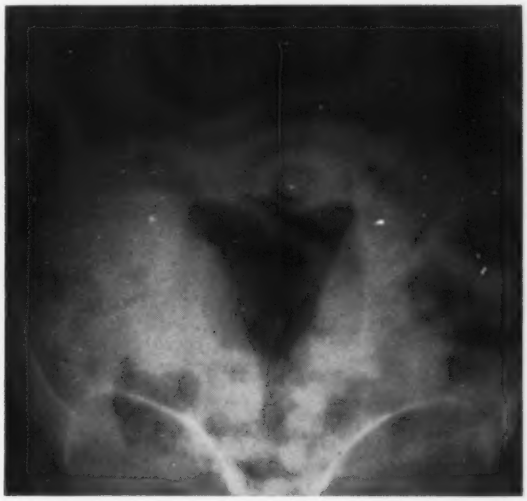

Fig. 8. Case V. Pneumoencephalogram in the anteroposterior view demonstrated the fan-shaped cavum veli interpositi. The fornices are depressed. Note the choroidal fissure on the right.

Case IV. R.W.M. Hospital No. 58-1918. The mother of this patient was being treated for epilepsy. The patient started to have daily generalized convulsions beginning on the second day of life. The convulsions could not be controlled with the usual anticonvulsive drugs and pyridoxin, yet stopped with diamox. Physical examination and psychomotor development up to the age of six months were normal. The spinal fluid was normal. Pneumoencephalograms revealed a cavum veli interpositi (Fig. 7) above and posterior to the third ventricle and a cisterna venae magnae cerebri separated from it by the vein of Galen. *Impression:* Cavum veli interpositi. Generalized convulsions.

Case V. R.C. Hospital No. 58-575. Both maternal grandparents died from cerebral hemorrhage. A brother of the mother has Friedreich's ataxia; one sibling had two convulsions. The patient's birth weight was 2,930 gm. He was admitted to the State University of Iowa Hospital at the age of four months because he did not thrive. There was a slight delay of psychomotor development. The body weight was 4,120 gm., height 57 cm., head circumference 40 cm. There was considerable muscular hypotonia with hyperactive reflexes; micrognathia, blue sclera, bilateral otitis media, and diastasis of abdominal recti. Blood chemistry and cerebrospinal fluid were normal. There was no evidence of any organic disease. Pneumoencephalograms revealed in the anteroposterior view (Fig. 8) the

FIG. 9. Case v. Pneumoencephalogram in the *left* lateral view demonstrates cavum veli interpositi, cisterna venae magnae cerebri and the band of radiodensity between the two produced by the vein of Galen.

cavum veli interpositi in the midline between the lateral ventricles. The choroidal fissure was well outlined on the right. The fornices were depressed from their more normal horizontal position. In the *left* lateral view (Fig. 9) the cavum was well seen. Air in the pericallosal sulcus gave evidence as to the thickness of the corpus callosum in this area. Figure 10 is a re-

FIG. 10. Case v. Retouched *right* lateral pneumoencephalogram shows the cavum and the cisterna venae magnae cerebri indented at the arrow by the vein of Galen.

FIG. 11. Case vi. Pneumoencephalogram in the lateral view demonstrates a cavum veli interpositi and evidence of cortical atrophy.

touched *right* lateral view to show structures more clearly. *Impression:* Cavum veli interpositi. Psychomotor retardation, growth failure, muscular hypotonia.

CASE VI. L.V. Hospital No. 57-15043. The family history, pregnancy and delivery in this case were normal. Birth weight was 3,420 gm. The child was admitted to the hospital for the first time at the age of four months and subsequently several other times because of anorexia and failure to gain weight, recurrent otitis media, and pneumonia. At the age of six months a patent ductus arteriosus was ligated. In addition, the anterior portion of a double aortic arch was severed to help release tracheal compression. On several occasions a reducing substance was found in the urine. This was not identified, but was not glucose or galactose. A low glucose tolerance test and a low value for gamma globulins were found. Gastrointestinal series, electroencephalogram, blood chemistry and cerebrospinal fluid were all normal. A pneumoencephalogram done at the age of nine and one-half months revealed a cavum veli interpositi (Fig. 11) above and posterior to the third ventricle. The anteroposterior view (Fig. 12) showed the cavum in the midline. It was fan-shaped, the upper curvilinear margin lying between the lateral ventricles. The right fornix was depressed from its normal position.

At eleven months the weight was 7,700 gm., height 66 cm., head circumference 46 cm. Although the child was normal neurologically,

Fig. 12. Case vi. Pneumoencephalogram in the anteroposterior view reveals the fan-shaped cavum in the midline and depression of the right fornix.

Fig. 13. Case vii. Pneumoencephalogram in the lateral view demonstrates a cavum veli interpositi, the outline of which could not be erased prior to making the print.

there was slight delay of psychomotor development. *Impression:* Cavum veli interpositi, double aortic arch, patent ductus arteriosus, delay of growth and psychomotor development.

Case vii. C.W.B. Hospital No. 57-16795. The mother of this patient had uterine hemorrhages during the first three months of pregnancy. The patient was admitted at the age of nineteen months because of delayed psychomotor development. The weight was 7,700 gm., height 70 cm., head circumference 41 cm. Roving nystagmus was present. There was a failure of macular development with probable poor central vision. Severe muscular hypotonia, active deep tendon reflexes, positive pyramidal signs were also noted. The electroencephalogram was normal. Pneumoencephalograms revealed a cavum veli interpositi above and posterior to the third ventricle (Fig. 13). It was separated from the cisterna venae magnae cerebri by the vein of Galen. In its lower anterior portion the quadrigeminal plate could be seen indenting the air column. The ventricles showed moderate dilatation. Figure 14 is retouched to show the structures more clearly. *Impression:* Cavum veli interpositi, microcephaly, psychomotor retardation, amblyopia.

Case viii. P. C. Hospital No. 58-6616. This patient was born six weeks prematurely with a birth weight of 2,260 gm. Psychomotor development was normal; he was toilet trained at two and one-half years of age. Between the age of three and three and one-half years, the child had 7 right-sided hemiconvulsions. The patient is left-handed. There were no abnormal neurologic findings. The electroencephalogram re-

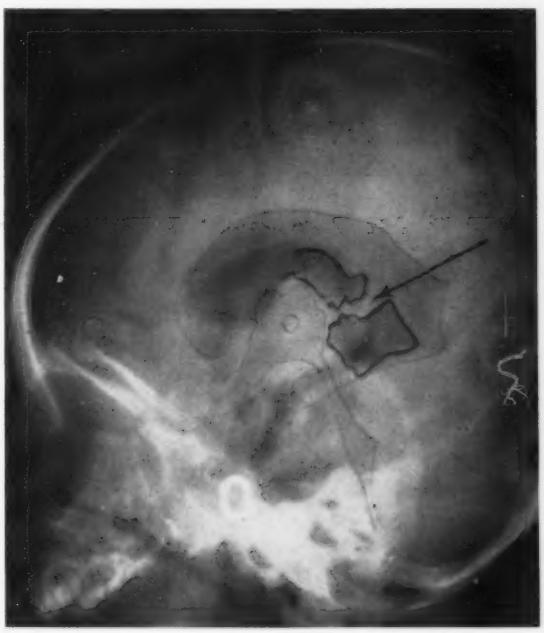

Fig. 14. Case vii. Retouched lateral pneumoencephalogram of Figure 13.

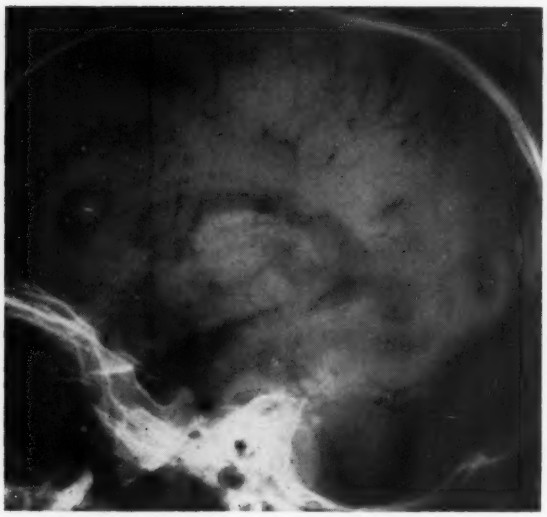

FIG. 15. Case VIII. Pneumoencephalogram in the lateral view shows a better filled cavum veli interpositi than third ventricle. It appears to arise from the lateral ventricle but does not.

vealed generalized spikes and slow left frontotemporal activity. A pneumoencephalogram demonstrated a cavum veli interpositi in the lateral view (Fig. 15) better filled than the third ventricle. In the anteroposterior view (Fig. 16) the cavum did not extend as high as many but lay above the third ventricle. The fornices were horizontal. A left carotid arteriogram revealed findings compatible with a space consuming lesion in the left temporal region.

FIG. 16. Case VIII. Pneumoencephalogram in the anteroposterior view shows horizontal fornices and the cavum in a lower position between the ventricles than usual.

FIG. 17. Case IX. Ventriculogram with air in subarachnoid space reveals a small poorly defined midline cavum veli interpositi above and posterior to the third ventricle.

A craniotomy failed to reveal such a lesion. *Impression:* Cavum veli interpositi, prematurity, right-sided hemiconvulsions.

CASE IX. B.S., aged forty-five years. Hospital No. 56-6887. This patient began to have a staggering gait at the age of forty-two years, was not able to focus his eyes, became clumsy and irritable. Physical examination revealed a vertical and horizontal nystagmus. Eye grounds were normal. The Babinski sign was positive, finger-nose and knee-heel tests indicated ataxia. The patient had an ataxic gait, could not squat nor stand on heels or toes and had a positive Romberg sign. The cerebrospinal fluid was clear with normal initial pressure, no cells, and proteins of 49 mg. per cent. Ventriculograms demonstrated air in the subarachnoid space (Fig. 17). There was a small, irregular collection of air above and posterior to the third ventricle. In the posteroanterior view (Fig. 18 *A*) there was noted some depression of the fornices and the small cavum. Figure 18 *B* shows the frontal sinus is superimposed simulating a cavum veli interpositi. *Impression:* Small cavum veli interpositi. Multiple sclerosis.

DISCUSSION

Analysis of our cases as well as those collected from the literature reveals a wide variety of symptoms. The great majority of the cases showed mental retardation, occa-

Fig. 18. Case IX. (*A*) The cavum is small, midline and there is some depression of the fornices. (*B*) The frontal sinus simulates the cavum.

sional idiocy, and, at times, normal intelligence. Case IX was normal until the onset of his multiple sclerosis and Cases II and VIII, whom we were able to follow beyond the age of one year, revealed a normal psychomotor development. Convulsions were present in at least two-thirds of the cases reported and in 4 cases of this present series. Some convulsions were quite resistant to therapy. Spastic phenomena were reported in several cases, yet three of our cases were characterized by considerable muscular hypotonia and one had congenital muscular hypertrophy. Pronounced hydrocephalus was never found, but both microcephaly and macrocephaly have been described. Associated conditions such as diffuse sclerosis, multiple sclerosis and spina bifida have also been described. Schönenberg[15] found pseudoatrophy of the optic nerve in 3 cases, which he interpreted as being a manifestation of defective myelinization. Zellweger[20] found 2 cases of amaurosis and optic atrophy, but did not define it more accurately. Our Case VII had a developmental failure of the macula. Dyschondroplasia,[15] craniofacial dysostosis[20] and Ollier's disease[18] have been reported. The combination of constitutional bone disease with cavum veli interpositi is quite interesting, although no further comments are possible at this time.

It is apparent that we cannot relate a single symptom revealing the presence of a cavum veli interpositi. It certainly is not responsible for the clinical symptoms. In some cases it might be associated with other malformations which in turn may be accountable for some of the clinical findings.

As mentioned previously, the tela choroidea of the third ventricle is originally a widely open pocket between two layers of pia mater. This later becomes a narrow choroid or transverse fissure when the commissural midline structures grow caudad. Instead of this being an open cistern, it is a potential one which can be opened by distortion in growth pattern or rate of growth, or by disease or tumor involving the surrounding structures, particularly the corpus callosum. This structure which reaches its final longitudinal extension by the end of the fifth fetal month has not yet reached its final thickness, since many fibers connect-

ing the two hemispheres develop later on. This could then explain why almost 75 per cent of the reported cases occur at a very early age, *i.e.*, in the first year of life. The persistence of the cavum veli interpositi may have different causes: (1) The cavum may persist if the pressure of the neighboring structures is diminished, such as might occur in a case of hypoplasia of the corpus callosum. In 2 of Zellweger's cases autopsy findings included hypoplasia of the corpus callosum. (2) It is conceivable that the pressure of neighboring structures decreases in some cases, as in the case of widespread cerebral phlebothrombosis with production of encephalomalacia involving particularly the posterior portion of the corpus callosum.[20] (3) Finally, the cavum may reopen as was demonstrated by Kruse and Schaetz. In their case of tuberculoma the quadrigeminal plate pushed the splenium of the corpus callosum upwards. In another case an angiomatous lesion of the choroid plexus widened the space of the velum interpositum.[8]

SUMMARY AND CONCLUSIONS

1. Nine new cases of visualization of the cavum veli interpositi are presented. The embryologic development, the roentgenologic appearance, and a theory as to its presence primarily in children under one year of age are discussed.

2. Forty-two other cases collected from the literature are analyzed, in addition to our 9 cases, from a clinical standpoint with described autopsy findings if they are available.

3. Cavum veli interpositi is a part of the subarachnoidal system.

4. It lies above and posterior to the third ventricle and may be of varying size and shape.

5. It occurs frequently in mentally retarded children but is not the cause of the retardation.

6. Its presence is frequently associated with abnormality in development or disease in or around the corpus callosum.

7. It is frequently confused with, but usually can be differentiated from, a cavum Vergae.

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CALCIFICATION OF THE BASAL GANGLIA OF THE BRAIN*

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PHYSIOLOGIC intracranial calcification visible on roentgen examination of the skull is common, being seen most often in the pineal gland and less frequently in the choroid plexuses and falx cerebri.

Less commonly, abnormal intracranial calcific deposits are seen on roentgenograms of the cranial vault. The etiologic factors responsible for such abnormal calcifications are legion and include degenerative changes secondary to trauma, infection, infestation, congenital abnormalities, new growth, metabolic disorders, aging, and unknown factors.

Rarely, the roentgen-visible intracranial calcifications are bilateral, symmetric, and located in the basal ganglia of the brain, frequently with concomitant calcific deposits in the dentate nuclei of the cerebellum and/or other regions of the brain.

This paper includes case reports of a group of patients who, during roentgenographic examination of the skull, were found to have calcification of the basal ganglia. It attempts to illustrate a portion of the spectrum of disease processes, many still incompletely understood, in which this rare calcification has been observed to occur. For the most part the patients were seen at The National Hospital, Queen Square, London, England.

HYPOPARATHYROIDISM

Hypoparathyroidism is an uncommon but well studied clinical and biochemical syndrome characterized by hypocalcemia, hyperphosphatemia, and tetany or tetanic equivalents. Frequently, epileptiform attacks, intellectual and psychotic changes, and ectodermal defects, including cataracts, may be present. The hypoparathyroidism may be idiopathic, but is more often a complication of thyroid surgery.

Both types show a positive response to the administration of parathyroid extract (Ellsworth-Howard test) with a phosphorus diuresis and elevation in serum calcium.[10] Treatment with vitamin D (or A.T.-10) with or without additional oral calcium usually produces a favorable symptomatic and biochemical response.

The occurrence of intracranial calcification in patients with chronic idiopathic and postoperative hypoparathyroidism is well known; this calcification occurs not only in the basal ganglia, but also in the dentate nuclei of the cerebellum and/or other areas of the brain.[6,17]

Five patients with idiopathic hypoparathyroidism and calcification in the basal ganglia have been seen in The National Hospital since 1950, all having been referred to the hospital for investigation of symptoms referable to the nervous system. Figure 1, *A*, *B*, *C*, *D*, *E* and *F* demonstrates the position, character, and distribution of this calcification on lateral skull roentgenograms of these 5 patients and an additional patient with idiopathic hypoparathyroidism who was treated at another hospital. All 6 patients showed the typical biochemical changes seen in hypoparathyrodism, with the lowest pretreatment serum calcium values ranging from 5.0 mg. per cent to 7.0 mg. per cent and the highest pretreatment serum phosphorus values ranging from 3.6 mg. per cent to 8.4 mg. per cent. Detailed clinical histories on 4 of these patients and reproductions of the roentgenograms of one have been published elsewhere.[23,24] Brief clinical histories of the remaining 2 patients follow.

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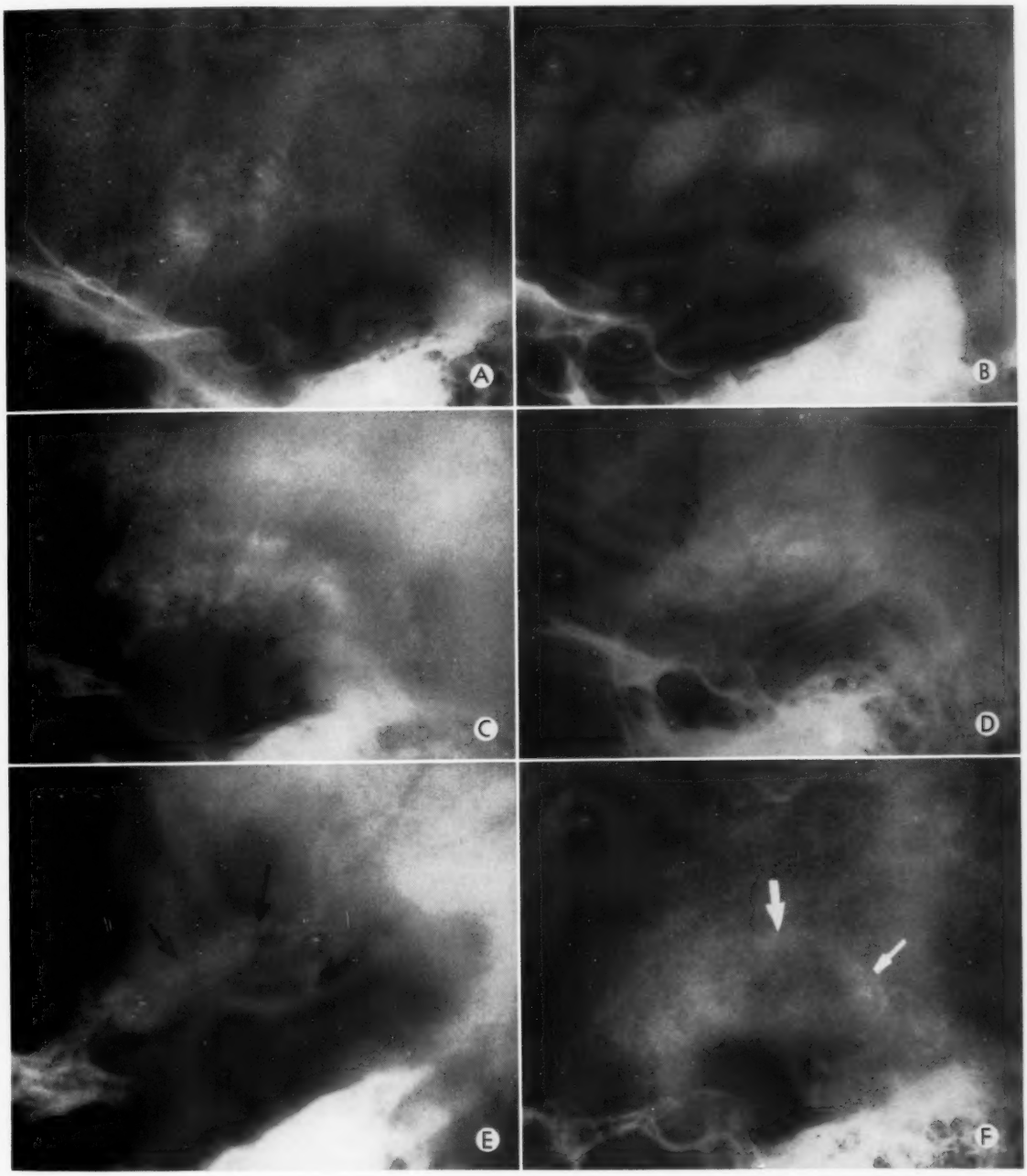


Fig. 1. (*A–F*) Calcification in the basal ganglia of 6 patients with chronic idiopathic hypoparathyroidism. Note the position of this abnormal calcification, considerably anterior to the physiologic calcification occasionally seen in the choroid plexuses. The radiating pattern of some of the calcific deposits in *A* and *B* suggests that some of the calcification is in gray matter between fibers of the internal capsule. (*B*) Case 1. (*F*) Case 11.

REPORT OF CASES

Case 1. E.K., No. 25972, a forty-four year old white male, was referred to The National Hospital in 1950 because of "fits" resembling petit mal attacks, progressive dulling of mentality, and deterioration of social habits, all of three years' duration.

On examination the patient was found to be unkempt in appearance, affable, but easily irritated. He had a mild dysarthria, showed a

moderate degree of dementia, and had bilateral cataracts.

Serum calcium was found to be 5.4 mg. per cent and serum phosphorus 6.3 mg. per cent. An electroencephalogram showed an abnormal rhythm suggesting a deep central cause, and a lesion of the basal nuclei or brainstem was considered possible.

Roentgenograms of the skull (Fig. 1*B*) revealed bilateral symmetric calcification, evidently in the head and body of the caudate nucleus.

The patient was transferred to Guy's Hospital under the care of Dr. M. J. McArdle where the diagnosis of idiopathic hypoparathyroidism was confirmed, and the patient was placed on calciferol therapy. He was last seen at Guy's hospital in September, 1957, at which time he was essentially asymptomatic.

CASE II. E.B., No. A45210, a forty-two year old white male, was referred to Guy's Hospital in March, 1955, under the care of Sir Charles Symonds and Dr. M. J. McArdle because of nocturnal "fits" of twenty years' duration and mental retardation of two years' duration.

On examination he was found to be of pleasant humor, slow in speech, and had a moderate impairment of mental ability. There were bilateral lens opacities, both optic disks were blurred, but there was no papilledema. Reflexes, although sluggish, were present, and there was a positive Chvostek and Trousseau sign.

Serum calcium was 6.0 mg. per cent and serum phosphorus 3.6 mg. per cent. An electroencephalogram revealed a widespread abnormality without constant focal distribution.

Roentgenograms of the skull showed faint calcification in the basal ganglia and dentate nuclei. There were also calcific flecks in the parietal regions of the brain (Fig. 1*F*).

A diagnosis of idiopathic hypoparathyroidism was made, and the patient was placed on oral calciferol therapy. He showed a gratifying return of mental function to premorbid levels and became free of nocturnal attacks. He was last seen in January, 1958, at which time he was well and had resumed the conduct of his business. His last serum calcium determination was 10.2 mg. per cent.

PSEUDOHYPOPARATHYROIDISM AND PSEUDO-PSEUDOHYPOPARATHYROIDISM

1. *Pseudohypoparathyroidism.* This syndrome was first described by Albright *et al.*[1] in 1942. Patients with this disease show the clinical picture of hypoparathyroidism, as previously described, with low serum calcium, high serum phosphorus, manifestations of tetany, and frequently ectodermal defects and intellectual disturbances.

However, unlike patients with true hypoparathyroidism, those with pseudohypoparathyroidism show a negative Ellsworth-Howard test, with neither a phosphorus diuresis nor a rise in serum calcium following administration of parathyroid extract. Both the hypoparathyroid biochemistries and the failure to respond to parathyroid extract are considered to be due to failure of the end organs of the body to respond normally, both to the patient's own hormone and to parenteral parathyroid extract, rather than a lack of endogenous hormone.

Although available commercial preparations of parathyroid extract are not always of reliable potency, and evidence is available which suggests the possibility that more than one active agent may be present in the extracts,[9] the Ellsworth-Howard test is still generally regarded as an important criterion in establishing the diagnosis of pseudohypoparathyroidism. The test, however, has not given consistent results, and its validity has recently been questioned.[13]

In addition to their negative response to administered parathyroid extract, patients with pseudohypoparathyroidism show other conspicuous physical and roentgenologic features which help to distinguish them from patients with true hypoparathyroidism. Typically, they are short in stature, with thick-set figures, and rounded facies. Shortening of the metacarpals and metatarsals and ectopic soft tissue calcification and ossification are frequent albeit inconstant findings.

Although the patients do not respond to parathyroid extract, they do respond to vitamin D (or A.T.-10) and oral calcium therapy in much the same manner as patients with true hypoparathyroidism; however, larger doses are sometimes required.

2. *Pseudo-pseudohypoparathyroidism.* In

1952 Albright and his colleagues[2] introduced this name to describe the clinical and biochemical findings in a twenty-nine year old female who showed many of the physical and roentgenologic characteristics seen in pseudohypoparathyroidism, as described above, but who showed no manifestations suggesting hypoparathyroidism and had normal blood calcium and phosphorus levels. Since 1952 a small number of additional cases of this entity have been reported.[16,18,25,27,37] It is of interest to note that several of these patients had blue sclerae, but no evidence of osteogenesis imperfecta.

Abnormal calcium deposits in the brain, including the basal ganglia and dentate nuclei, are common in pseudohypoparathyroidism, but intracranial calcification has not yet been reported in pseudo-pseudohypoparathyroidism.

Case III,* known to have pseudohypoparathyroidism in the past, is consequently of interest both roentgenologically and endocrinologically. Within the period that she had been under medical surveillance, calcification in her basal ganglia had developed. During most of this same period the patient received no specific therapy to correct her hypoparathyroid biochemistries. In spite of this, the patient's abnormally low blood calcium and abnormally high blood phosphorus have reverted to essentially normal values. In effect, the patient appears to have converted spontaneously from the status of pseudohypoparathyroidism to that of pseudo-pseudohypoparathyroidism,[8] while during the same period roentgenographically visible calcification in the basal ganglia has developed.

REPORT OF A CASE

Case III. E.M., No. AD575, a twenty-year old single white female, was admitted to University College Hospital, London, in June 1957, under the care of Professor C. E. Dent, for investigation of anorexia, nausea, and abdominal pain of recent onset. The patient had been investigated in Canada some eleven years previously and was found to have pseudohypoparathyroidism.

The patient had been a full-term baby with a birth weight of $8\frac{1}{2}$ pounds. She had walked at twelve months of age and talked at two and one-half years of age. She had four living siblings, all of whom were in good health and showed none of the usual stigmata of pseudohypoparathyroidism.

At about the age of seven she had convulsions following a cold. In 1946, at the age of eight years and nine months, she was investigated in Canada under the care of Dr. J. S. L. Browne because of "epilepsy," mental retardation, lower abdominal pain, and leg cramps. At that time she was found to have the clinical and biochemical features of pseudohypoparathyroidism. She was short (4 feet $1\frac{3}{4}$ inches) and obese with a round face and flattened nosebridge. Her extremities were relatively short; there was metacarpal shortening and soft tissue calcification, and she showed a marked genu valgum. The Chvostek sign was markedly positive, serum calcium was 4.5 mg. per cent, and serum inorganic phosphorus 10.0 mg. per cent. Roentgenograms of the skull were normal and showed no calcification in the basal ganglia.

There was no clinical response to parathyroid hormone therapy. She was placed on A.T.-10 and calcium gluconate with resultant disappearance of her Chvostek sign, improvement in her previously abnormal electroencephalogram, and return of her abnormal biochemistries to a normal serum calcium of 10.6 mg. per cent and a serum phosphorus of 6.5 mg. per cent.

There is no indication that the patient adhered to her prescribed therapeutic program once she left the hospital in Canada, and she was next seen at the Hospital for Sick Children, London, in July, 1947, at the age of nine years and nine months, because of "fits," cramps, and pain in the left shoulder. She was found to have a positive Chvostek sign, abnormally low serum calcium, and abnormally high serum phosphorus. Roentgenograms of the skull again showed no evidence of basal ganglia calcification (Fig. 2, *A* and *C*). A.T.-10 and calcium gluconate therapy was instituted, but in September, 1947, she showed symptoms of hypercalcemia. Her serum calcium was found to be 16.7 mg. per cent and therefore the A.T.-10 and calcium therapy was discontinued.

* Albright and Reifenstein[3] in 1948 and Selye[26] in 1949 have both referred briefly to this patient and to her mother, but the complete history has never been reported.

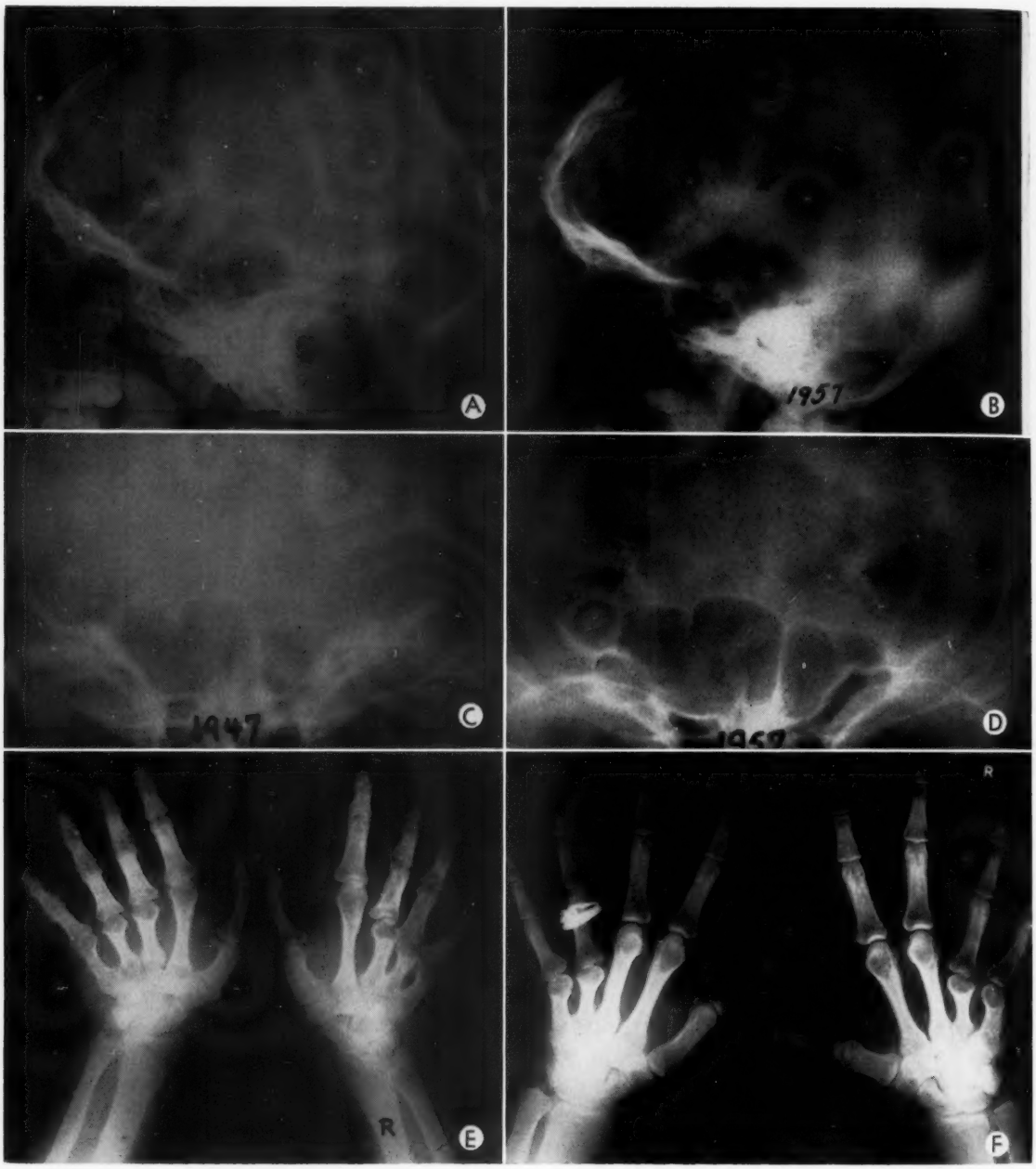


FIG. 2. Case III. (*A*) Lateral skull roentgenogram taken in 1947 shows no evidence of basal ganglia calcification. (*B*) Ten years later. Calcification in the basal ganglia is now visible (arrows). (*C*) Anteroposterior view of the skull taken in 1947; there is no evidence of intracranial calcification. (*D*) Ten years later. Symmetric calcium deposits in the basal ganglia are now evident (arrows). (*E*) Hands showing shortened metacarpals and ectopic soft tissue calcifications. (*F*) Mother of Case III. Hands showing similar shortening of some of the metacarpals and calcifications in the soft tissues. There is also shortening of some of the terminal phalanges.

She was subsequently seen in Edinburgh, Scotland, in December, 1947, at the age of ten years and three months, at which time her calcium was 11.2 mg. per cent and phosphorus 3.6 mg. per cent. Three months later blood determinations showed her serum calcium to be 9.2 mg. per cent and her serum phosphorus 4.8 mg. per cent.

She was next seen at the University College of the West Indies in February, 1955, at the

age of eighteen years and five months, seven and one-half years after discontinuance of any specific therapy. Here serum calcium was found to be 8.8 mg. per cent; her phosphorus 7.0 mg. per cent; blood urea 17 mg. per cent; and her basal metabolic rate −24 and −27 per cent. She was placed on calciferol, calcium lactate, and thyroxin, but in April, 1956, all but her thyroxin were discontinued because of vomiting, hypertension, and albuminuria.

In June, 1957, at the age of twenty years and nine months, she was referred to Professor C. E. Dent. At that time her only medication was thyroxin. The Chvostek sign was positive, her serum calcium was slightly low at 8.5 mg. per cent, and her serum phosphorus essentially normal at 4.6 mg. per cent. Several days before admission to University College Hospital in June, 1957, she was started on moderate daily dosages of vitamin D. Concomitant with the vitamin D administration, the patient experienced nausea, anorexia, and abdominal pain. Although the patient's symptoms were felt to be coincident with rather than consequent upon the vitamin D administration, she was admitted for observation. At the time of admission she was in no acute distress.

Pertinent findings on examination at that time were as follows: she was a short (4 feet $8\frac{1}{2}$ inches), obese, childish female with a round face, flat nasal bridge, and short neck. Her limbs were short, hands tiny and held in slight ulnar deviation, and there was metacarpal shortening with the index finger of both hands longer than the other fingers. A 7-inch separation of the internal malleoli was present when the thighs were held adducted. An alternating external strabismus was evident, but no lenticular opacities could be seen, nor were blue sclerae reported. Body hair was sparse but of normal distribution. There was no breast development. The patient had menstruated only once six years previously. Blood pressure was 130/100, and palpation of the abdomen revealed a generalized tenderness.

The urine contained a small amount of protein and some pus cells; there was no aminoaciduria, a normal "glycine" pattern was present, and the calcium concentration in the urine was low.

Serum calcium was 9.7 mg. per cent and blood urea 24 mg. per cent.

Roentgen examinations showed the presence of calcification in the basal ganglia (Fig. 2, *B* and *D*), shortening of all but the second metacarpals (Fig. 2*E*), scattered soft tissue calcifications, most of which were in the extremities, a punched-out erosion in the third right proximal manual phalanx, exostoses at the upper ends of both tibiae and on the right ulna, and an apparent chondroma of the right scapula. The second metatarsal on the left was conspicuously longer than the corresponding metatarsal on the right.

The patient's hospital course was uneventful. No evidence of hypercalcemia had been found, the abdominal pain subsided without specific treatment, and she was discharged on A.T.-10 therapy. She had been taking thyroxin for several years, and this was continued during the above treatment so that the effect of only one change of therapy could be determined. Her last blood determinations on July 15, 1957, showed a serum calcium of 9.3 mg. per cent and a serum phosphorus of 3.6 mg. per cent.

While the patient was being studied in Canada in 1946, it was noted that her mother had shortened metacarpals, soft tissue calcification, tibial spurring, and bowing of the radii. She did not, however, have any of the bodily or facial appearances common in pseudohypoparathyroidism, and there had been no history of previous illness or tetany. She was found to have a slightly positive Chvostek sign; serum calcium was normal at 10.8 mg. per cent, but her serum phosphorus was elevated at 6.1 mg. per cent.

The mother has remained asymptomatic over the years, and except for the shortened metacarpals and soft tissue calcifications (Fig. 2*F*) evidences none of the clinical stigmata of pseudohypoparathyroidism. Her chemistries in July, 1957, were normal with serum calcium 9.6 mg. per cent and serum phosphorus 3.9 mg. per cent. Selye[26] in 1949 referred to her briefly and considered her a mild form of pseudohypoparathyroidism, but, in the light of Albright and co-workers'[2] subsequent work published in 1952, it would appear that she is a case of pseudo-pseudohypoparathyroidism.

FAMILIAL BASAL GANGLIA CALCIFICATION

Roentgenographically visible calcification in the basal ganglia has beeen known to occur in families, manifested not only in siblings but in members of more than one generation of the same family.

In some of the instances reported, the patients have had pseudohypoparathyroid-

ism.[14] In other reported cases one or more of the affected members of the family suffered from neurologic and/or mental abnormalities but had no disturbances in blood calcium and phosphorus levels as measured by routine laboratory methods.[12,15,30]

The following case summaries illustrate instances of calcification in the basal ganglia detected in more than one individual of a family unit. In none of the patients cited were there any external physical characteristics suggesting either pseudohypoparathyroidism or pseudo-pseudohypoparathyroidism.

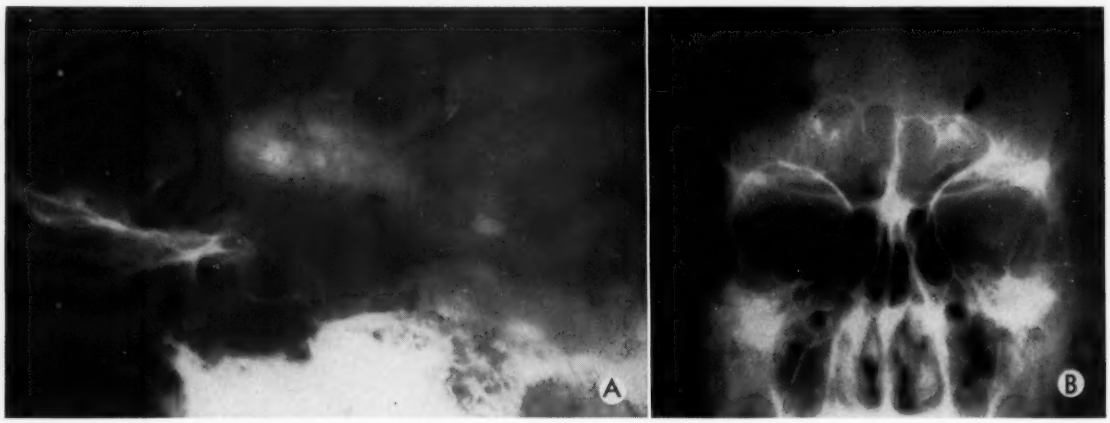
REPORT OF CASES

CASE IV. R.D., No. 22264, a forty-three year old white male, was first admitted to The National Hospital in 1950 under the care of Mr. Harvey Jackson. He had been subject to "fits" since childhood, his attacks being controlled by conservative therapy. Since the age of twenty-six, however, he had suffered several major convulsions each year, and during the last year had experienced occasional clumsiness and paresthesias in the left arm and leg. He was essentially asymptomatic on admission.

On examination the only significant abnormalities found were slow activity in his electroencephalogram without focal signs and dense calcification in the basal ganglia (Fig. 3, *A* and *B*). Blood calcium, phosphorus, and alkaline phosphatase were all normal. He was discharged without specific diagnosis and remained free of convulsions on luminal therapy.

He was next admitted to The National Hospital in 1954 because of progressively severe headaches, sometimes accompanied by vomiting. He felt that of late his memory had been poor, and he stated that three days before admission his speech had become somewhat slurred.

Again no significant abnormalities were found on physical examination or routine biochemical studies. Psychologic and psychiatric



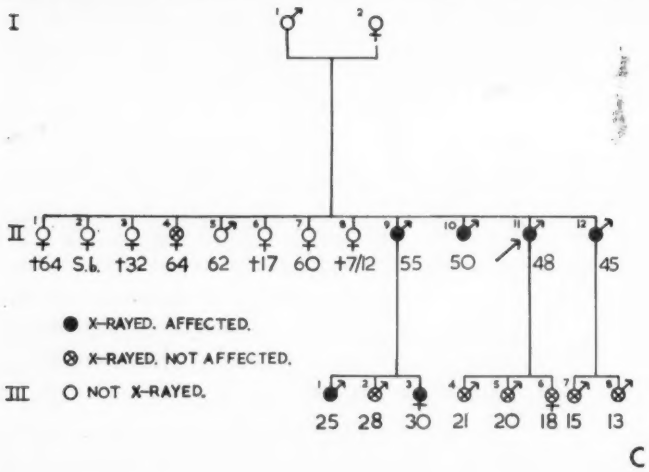


FIG. 3. Case IV. (*A* and *B*) Note the conglomerate character of the calcifications in the basal ganglia. (*C*) "Family tree" of Case IV. Long arrow (II-11) indicates the patient. His niece and nephew are III-3 and III-1, respectively. A cross before an individual's age indicates age at time of death which had occurred previous to the investigation; S.b. designates a stillbirth.

tests suggested a definite mild impairment of intellect from a high-average "premorbid" level.

At the suggestion of Mr. P. Carey, roentgenograms of the skull were taken of 12 other members of the patient's family, and three brothers, a niece, and a nephew were all found to have roentgen-visible calcification of the basal ganglia (Fig. 3*C*). All except the niece were asymptomatic.

The patient and the affected niece were subsequently hospitalized and careful balance studies performed by Dr. P. D. Roberts.[22] On repeated determinations the patient's blood calcium and phosphorus were found to be within normal limits except for several serum calcium values of 8.6 mg. per cent. He did, however, show evidence of altered phosphorus metabolism characterized by retention of phosphorus and a strongly positive phosphorus balance on a normal phosphorus diet. In addition, he had an abnormal response to intravenous calcium infusion similar to that seen in hypoparathyroidism, and he showed only a suboptimal phosphorus diuresis in response to intravenous parathyroid extract (Ellsworth-Howard test). The results were insufficient to establish a diagnosis of hypoparathyroidism. The patient was placed on A.T.-10 and later on calciferol therapy and, when last seen in 1957, had been free of convulsions and headaches.

The affected niece (Fig 3*C*, III-3) had a history of mental depression for which she had received electroshock treatment. Her blood calcium and phosphorus levels were normal, but her balance studies gave results similar to the patient R.D., with evidence of phosphorus retention and a strongly positive phosphorus balance, a suboptimal phosphorus diuresis in response to the Ellsworth-Howard test, and an equivocal response to intravenous calcium infusion.

The affected nephew (Fig. 3*C*, III-1) was examined at The National Hospital early in 1957 because of frequent severe headaches which allegedly followed a head injury sustained in a parachute jump. Except for the calcification in the basal ganglia, no abnormalities were found. Blood chemistries and electroencephalogram were normal, but balance studies were not performed. He was placed on calcium gluconate, and, when last seen in October, 1957, he was asymptomatic except for occasional headaches.

Case v.* J.M., No. 63139, a fifty-five year old white male, was admitted to The National Hospital in December, 1955, under the care of Mr. Harvey Jackson because of progressive memory loss, visual deterioration, weakness and involuntary movements of the right upper extremity, speech difficulties, and deafness on the right over a period of four months. Roentgenograms of the skull taken elsewhere had shown marked intracranial calcifications.

On examination the patient was confused, had dysphagia, a right-sided facial weakness, a right-sided spastic hemiplegia, and his fingers showed marked clubbing. There was a severe memory defect and a gross intellectual impairment. Blood calcium and phosphorus levels were normal.

Roentgenographic examination of the skull showed marked calcification in the basal ganglia and dentate nuclei of the cerebellum, with prominent medial displacement of the calcified basal ganglia on the patient's left (Fig. 4, *A* and *B*). Chest roentgenograms revealed opacities in the left upper lobe and in both hilar areas. Ventriculography confirmed the diagnosis of a space-occupying mass on the left, and biopsy revealed this mass to be a secondary carcinoma. Some sections taken at biopsy showed capillary calcification of gray matter.

The patient's condition progressively deteriorated, and he died in March, 1956. Postmortem examination revealed the lesion in the left chest to be a primary bronchogenic carcinoma, and the lesion in the left brain a large solitary metastasis. Gross calcareous deposits were found in the basal ganglia and dentate nuclei, most in the globus pallidus and putamen bilaterally.

Attempts to obtain skull roentgenograms of all members of the patient's family were unsuccessful, and only his married daughter appeared for examination. She, too, was found to have calcification in the basal ganglia of the brain (Fig. 4, *C* and *D*). The daughter was asymptomatic, and further clinical and biochemical examinations could not be obtained.

Case vi. A.P., No. 4479, a white male, was first admitted to the Queen Elizabeth Hospital for Children, London, in January, 1948, under the care of Dr. Helen Mackay. At that time he was fourteen years and nine months of age. His brother, aged nine, similarly but less se-

* A more detailed discussion of the findings in this unusual case has been reported by the authors in *Radiology*, 1959, *72*, 426.

verely affected with the same disease process, was admitted at the same time. The clinical histories and investigations performed on the two brothers up to that time have been reported by Neill and Dingwall,[20] and the interested reader is referred to their article for further details.

Briefly, the patient A.P. presented the following outstanding features: infantilism, microcephaly, retinal degeneration, mental deficiency deafness, hepato-splenomegaly, and intracranial calcifications, including calcification in the basal ganglia and dentate nuclei of the cerebellum (Fig. 5*A*). Serum calcium and phosphorus levels were normal. A diagnosis of atypical progeria was made.

The patient was subsequently re-admitted to the Queen Elizabeth Hospital for Children six times between 1950 and 1955, usually because of convulsions, wild behavior, or, as on his last admission, because of anorexia, weight loss, increasing number of "fits," vomiting, and incontinence.

The patient died in 1956 at the age of twenty-three. Postmortem roentgenograms demonstrated the marked calcifications in both cerebral hemispheres (Fig. 5*B*) and cerebellum (Fig. 5*C*). Neuropathologic studies were done by Dr. H. Urich[34] and colleagues, who will publish their findings in full in the near future. Briefly, the brain was very small and showed widespread demyelinization of the white matter and glial replacement of ventricular ependyma. Calcification appeared in several forms: arterial calcification seen in the centrum semiovale, the basal ganglia, and cerebellum; capillary calcification visible in the thalamus, cerebral and cerebellar cortex, the striatum, in-

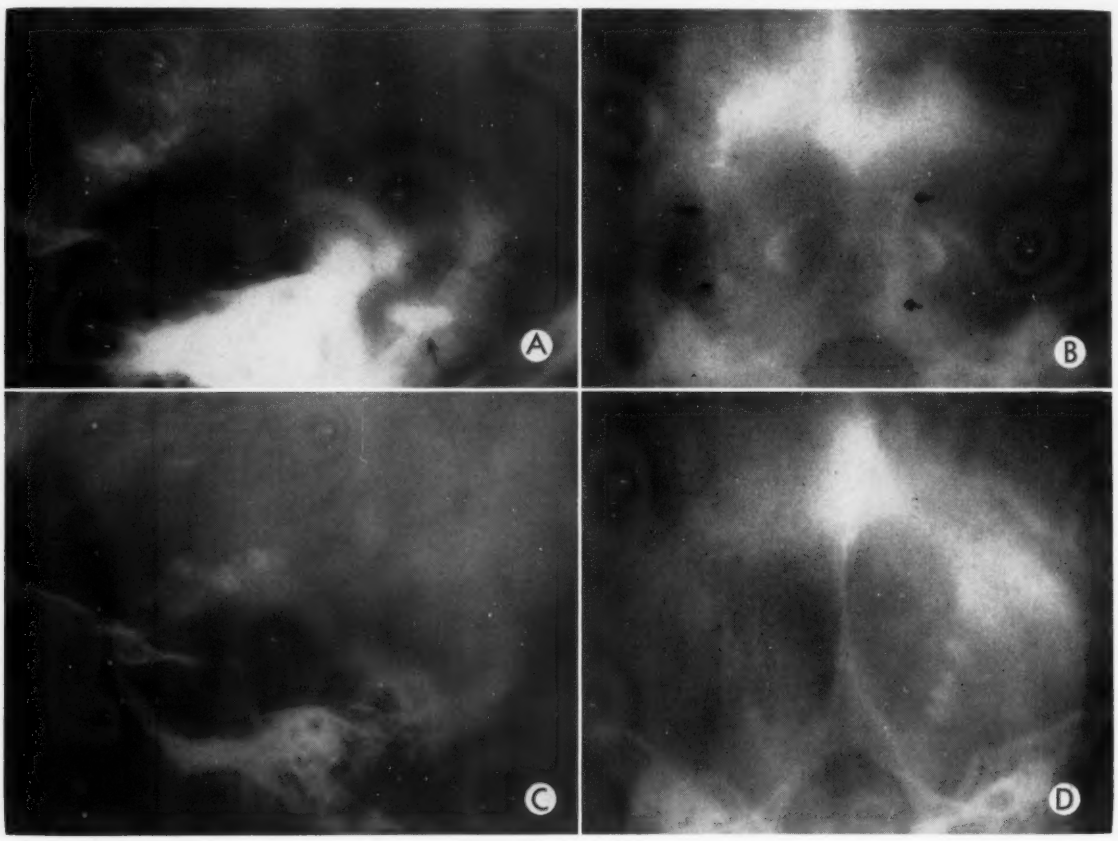


FIG. 4. Case v. (*A*) Note the radiating character of some of the calcification. The arrow points to dense calcification in the dentate nuclei of the cerebellum. (*B*) This view demonstrates the medial displacement of the calcified left basal ganglia. The more dense comma- and reversed-comma shaped calcifications are calcified dentate nuclei. (*C* and *D*) Comparable views of the skull of the patient's daughter. Note the scattered foci of calcification in the brain substance anterior and superior to the calcified basal ganglia. (Reproduced by permission from *Radiology*, 1959, *72*, 426).

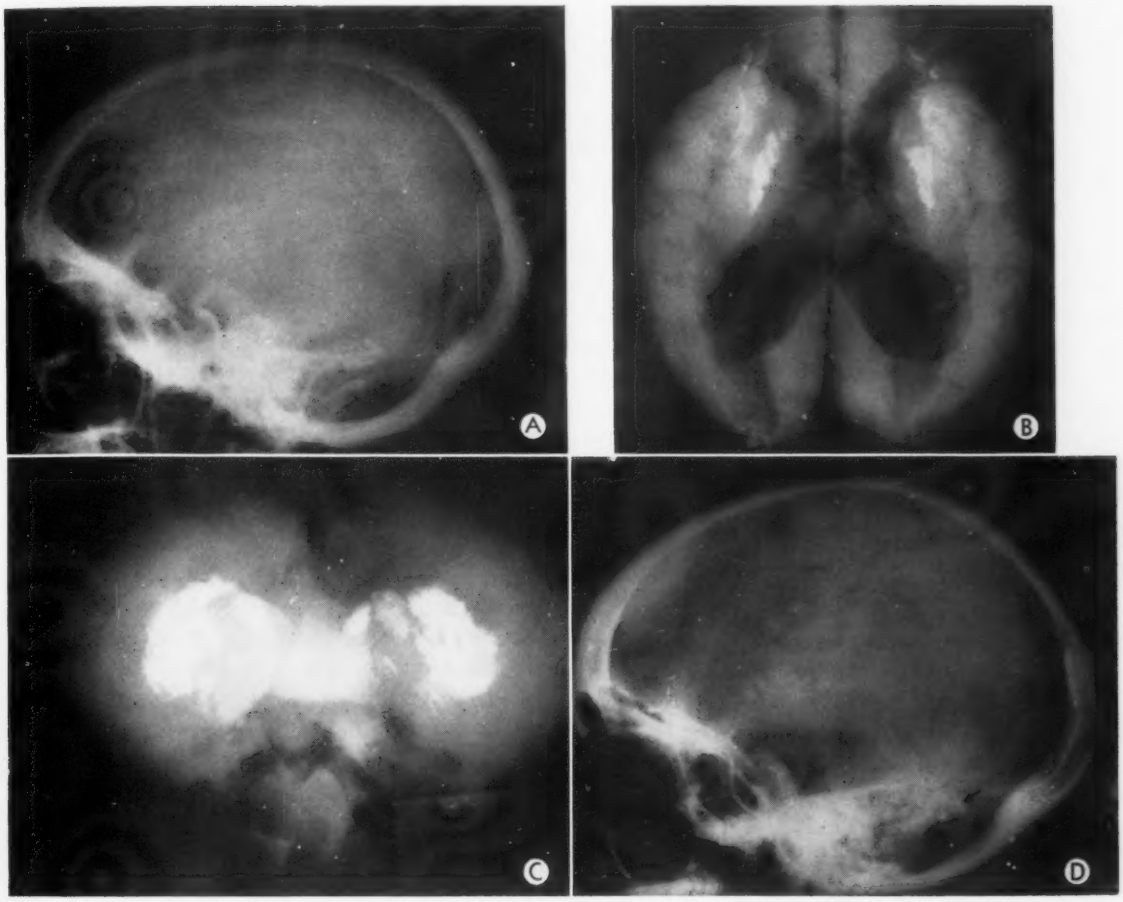


Fig. 5. Case vi. (*A*) In addition to the calcification in the basal ganglia and cerebellum, there is a considerable amount of cortical calcification. (*B*) Postmortem roentgenogram of the brain showing apparent symmetric dilatation of the ventricles and widespread calcifications. (*C*) Postmortem roentgenogram of the cerebellum demonstrating the dense calcification in the dentate nuclei as well as in other areas in both cerebellar hemispheres. (*D*) The younger brother of Case vi. There are no apparent cortical calcifications. The basal ganglia and dentate nuclei (arrow) are heavily impregnated with calcium.

ternal capsule, subthalamic region, and the red nucleus; loose calcium deposits in the shape of globules, dumb-bell forms, or in structures resembling a bunch of grapes in the cerebral cortex, striatum, pallidum, and cerebellum; and large concrements with concentrically laminated structure in the putamen, white matter of the cerebellum, and in some parts of the cerebral cortex.

The patient's younger brother was last admitted to the Queen Elizabeth Hospital for Children in 1953, and apparently is still alive. Roentgenograms of his skull showed considerable calcification in the basal ganglia and dentate nuclei of the cerebellum (Fig. 5*D*).

UNCLASSIFIED GROUP

The 3 following case summaries illustrate examples of bilateral symmetric calcification of the basal ganglia of patients in whom no evidence of clinical or biochemical hypoparathyroidism or pseudohypoparathyroidism was found. It is not known whether the families of the patients were examined, and it cannot be said with certainty that all other members of the families were free of roentgenographically visible intracranial calcification. Biochemical balance studies were performed only on the last of these 3 patients.

REPORT OF CASES

The first patient was considered clinically to be a case of toxoplasmosis although she lacked some of the usual stigmata, and microscopic evidence of the toxoplasma parasite was never found in the body fluids or tissues of the patient.

CASE VII.* M.B., No. 20065, a twenty year old white female, was admitted to The National Hospital in November, 1949, under the care of Dr. Macdonald Critchley. The patient had been mentally and physically retarded since infancy, and there had been lifelong recurrent gastrointestinal and febrile disturbances. Convulsions had occurred during the last eighteen months prior to admission.

On examination the patient appeared about the size of a twelve year old girl. There was no breast development or axillary hair and only scant pubic hair. Adequate examination was not possible, but the patient showed evidence of gross mental and visual impairment. There was no chorioretinitis. An electroencephalogram was grossly abnormal, resembling the record of a child three or four years of age, but without focal changes. Laboratory studies revealed a serum calcium of 11.2 mg. per cent, serum phosphorus 5.6 mg. per cent, elevated cerebrospinal fluid protein (100 mg. per cent), elevated erythrocyte sedimentation rate (34 mm./hour), and serum neutralizing antibodies to toxoplasmin were positive to a titre of 1/512. The patient's mother and a sibling showed no neutralizing antibodies.

Roentgenographic examinations showed approximately a two-year retardation in epiphyseal union and dense symmetric calcification of the basal ganglia, with several flecks and streaks of calcification scattered elsewhere in the brain substance (Fig. 6, *A* and *B*).

The patient died in 1950. Postmortem examination done at another hospital showed a mild degree of nephrocalcinosis, prepubertal uterus and adnexa, and very delayed maturation of the ovaries. Masses of calcification were visible in the basal ganglia on gross examination of the brain. Microscopically there was widespread calcification of vascular walls involving arterioles, venules, and even capillaries. This was sporadic in the cortex and cerebellum and most marked in the basal ganglia about the masses and plaques of calcification. There was marked neuronal loss in the basal ganglia with calcification of degenerated neurones. There is no mention either of the appearance of the parathyroid glands or of the identification of any toxoplasma organisms in the body tissues.

The second patient of this group presents a confusing clinical and roentgenologic picture. She was considered a case of "renal rickets," and at one period in her life was treated with insulin for an apparent diabetes.

CASE VIII. J.W., a ten year old white female,

* The clinical history of this patient prior to 1950 has been reported.[22] Death occurred in 1950, and postmortem findings can now be included in the present case summary.

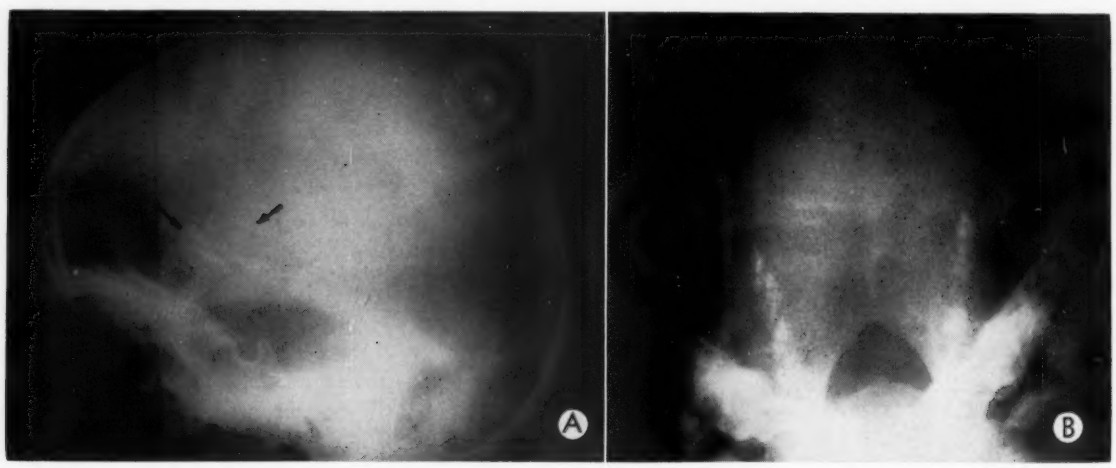


FIG. 6. Case VII. (*A*) The calcification in the basal ganglia is indicated by arrows. (*B*) Towne's view of the skull showing the marked calcification in both basal ganglia.

was admitted to the Princess Beatrice Hospital, London, in October, 1934, under the care of Dr. B. Buckley Sharp, because of difficulty in walking, pain in the back, polydipsia, and frequency of micturition.

On examination she was found to be short in stature for her age and had a marked genu valgum. Sugar and acetone were found in her urine, which contained many granular casts. Blood urea was 84 mg. per cent, serum calcium 13.0 and 10.0 mg. per cent on separate determinations, and serum phosphorus 3.2 mg. per cent. Glucose tolerance test showed a "blood sugar curve of mildly diabetic type."

Roentgenographic examinations were done at that time, but were subsequently lost or destroyed in the bombings in 1940. It is reported that they showed calcifications on the surface of the brain and in the falx. Numerous semiopaque deposits were seen throughout the renal tissues.

The patient was followed up from time to time at the Princess Beatrice Hospital until May, 1954, a period of almost twenty years. She was always regarded as a case of renal dwarfism and infantilism with renal rickets. The disturbance in sugar metabolism was treated initially with insulin, but later appeared to rectify itself, and insulin was discontinued. Last recorded blood chemistries during this period were in February, 1951, when her blood urea was 36 mg. per cent, serum calcium 11.0 mg. per cent, and fasting blood sugar 122 mg. per cent.

Late in 1954 she was seen at The National Hospital (No. 54284) because of severe headaches. Her blood calcium, phosphorus, and urea were found to be normal. Roentgenographic examinations revealed some asymmetry of the skull with extensive cortical calcification on the left side of the brain similar to that seen in the Sturge-Weber syndrome. There was also prominent calcification in the basal ganglia of the brain as well as in the dentate nuclei of the cerebellum (Fig. 7, *A*, *B* and *C*). There was bilateral renal calcinosis.

In 1955 she was transferred to another hospital for renal and metabolic studies. At that time she was found to be short in stature, with short extremities, a marked genu valgum, and moderate frontal "bossing" of the skull. There was obvious mental retardation and a severe myopia. Only a few carious teeth remained, all showing poor enamel and marked transverse ridging.

Examination of the patient's urine showed a 2 plus glycosuria, a negative reaction to the Sulkowitch test, and no proteinuria.

Fasting blood sugar, serum calcium, and serum phosphorus were normal.

In addition to the findings in the skull and kidneys, the radiologist felt that some slight "clubbing" of the ribs and ends of the bones might be due to a dyschondroplasia.

Unfortunately, the patient left the hospital against advice within twenty-four hours after admission, and no further studies were done. When last heard of in March, 1958, the patient was essentially bedridden. She was "aging" rapidly, continued to have severe headaches, and was troubled with menorrhagia. The patient was apparently unwilling to be readmitted to the hospital for any further diagnostic studies.

The last patient of this group, while being examined because of a congenital heart lesion, was found to have osteoporosis. During the course of investigations into the cause of her osteoporosis, calcification of the basal ganglia of the brain was discovered.

Case IX.* In June, 1957, J.W., a twelve year old white female, was referred by Dr. R. E. Bonham-Carter to University College Hospital, under the care of Professor C. E. Dent, for investigation of osteoporosis.

The child was known to have a ventricular septal defect, and her chief symptoms had been a persistent cough, frequent attacks of bronchitis, and dyspnea and cyanosis on exertion.

On examination she was found to be dwarfed and mentally retarded. Laboratory studies revealed a normal blood calcium and phosphorus. Roentgenographic examinations and calcium balance studies confirmed the diagnosis of osteoporosis, and roentgenograms of the skull disclosed symmetric calcification in the basal ganglia of the brain (Fig. 8, *A* and *B*).

No specific etiology for the calcification in the basal ganglia was determined. The child is being observed closely and further studies are contemplated.

* The complete clinical and biochemical findings on this patient will be published in full in the near future.[4] Only a very brief summary of features pertinent to the present discussion, therefore, is included herein.

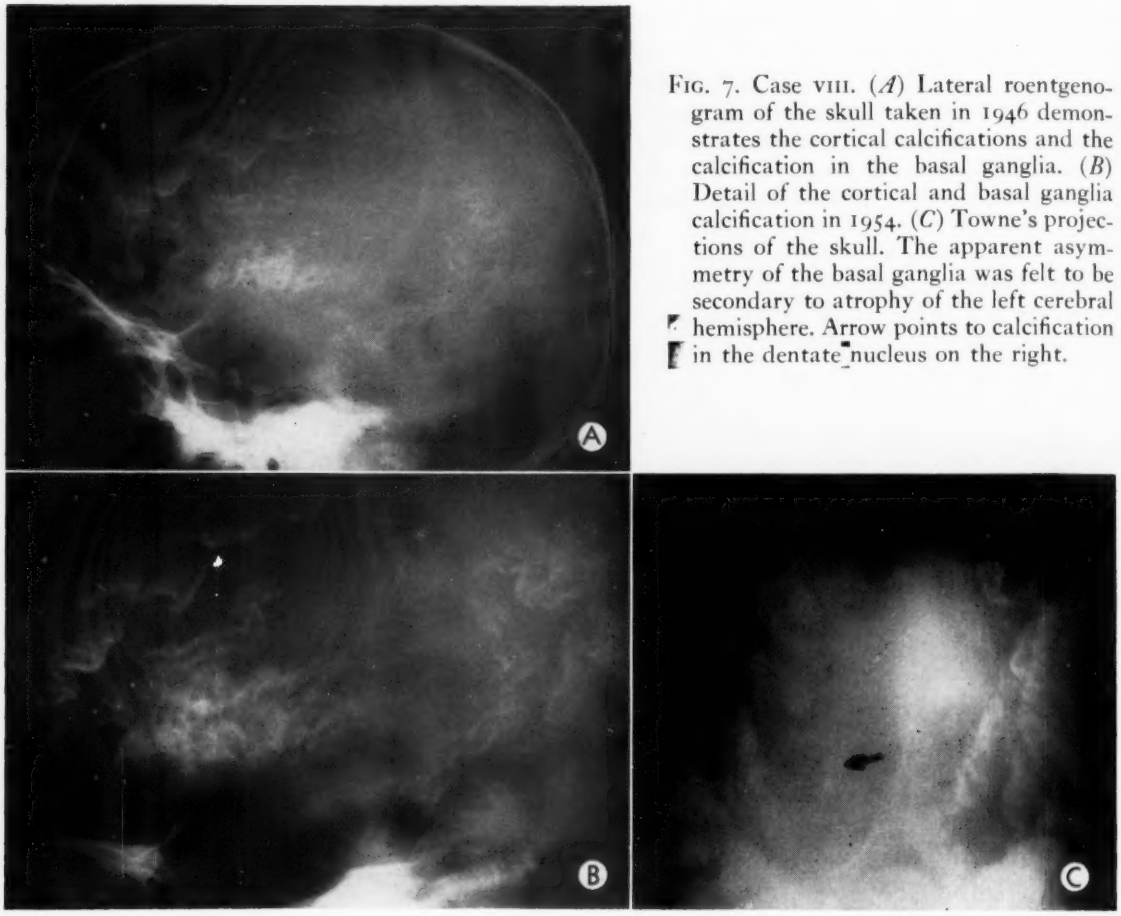


FIG. 7. Case VIII. (*A*) Lateral roentgenogram of the skull taken in 1946 demonstrates the cortical calcifications and the calcification in the basal ganglia. (*B*) Detail of the cortical and basal ganglia calcification in 1954. (*C*) Towne's projections of the skull. The apparent asymmetry of the basal ganglia was felt to be secondary to atrophy of the left cerebral hemisphere. Arrow points to calcification in the dentate nucleus on the right.

DISCUSSION

PATHOLOGY

Microscopic calcareous deposits in the basal ganglia of the brain have been shown to occur in a wide variety of disease states. They are also not unusual incidental findings in routine autopsy material.[29] On the other hand, gross calcification in the basal ganglia, visible on roentgenograms of the skull, is rare, and has been seen most fre-

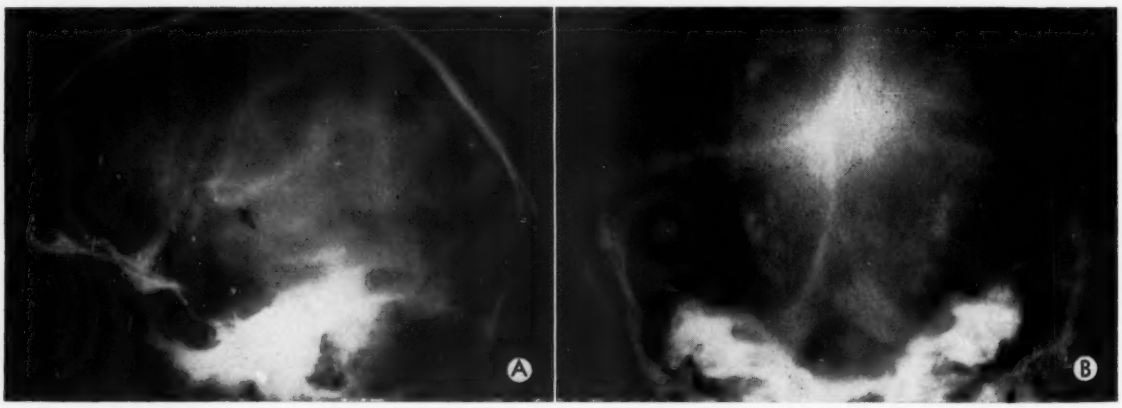


FIG. 8. Case IX. The arrow indicates the calcification in the basal ganglia. (*B*) The bilateral and symmetric character of the calcification as seen in the Towne's projection of the skull.

quently in chronic hypoparathyroidism, either idiopathic or postoperative, and in pseudohypoparathyroidism. In recent reviews about one-seventh of the cases of idiopathic hypoparathyroidism[29] and about a third of the patients with pseudohypoparathyroidism[13] showed such bilateral basal ganglia calcification.

The sequence of events in this calcification process apparently begins with the deposition of a colloid protein material in and about the walls of the smaller cerebral vessels. This substrate is subsequently impregnated first with iron and later with calcium, the latter collecting to form globules, sheaths, and concrements which may not only occlude capillaries, encase arterioles and venules, but can coalesce into large masses which obliterate completely the usual microscopic structures and become visible as large calcareous masses.

ETIOLOGY

The exact etiologic factor or factors responsible for these deposits in the basal ganglia, dentate nuclei, and other areas of the cerebral and cerebellar hemispheres are still obscure. However, after study of the published reports in the literature, it is difficult to avoid the speculation that the underlying causative factor is a transient or permanent dysfunction of parathyroid homeostatis due either to an inherited, developmental, or acquired defect.

1. *Hypoparathyroidism.* There is general agreement that the intracranial calcification in chronic hypoparathyroidism, both idiopathic and postoperative, is due to the altered calcium and phosphorus metabolism. However, beyond discussions of theoretic possibilities, no explanation, amenable to clinical or laboratory confirmation, has been advanced to explain the propensity of the basal ganglia and dentate nuclei for the accumulation of such large amounts of calcium.[17,19,21,24,36]

2. *Pseudohypoparathyroidism.* It is generally agreed that this syndrome represents a genetic disorder, probably involving several independent genes in variable degree, and producing several general types of abnormality: a disturbance in the patient's response to parathyroid hormone, a dyschondroplasia, and a strong tendency to develop ectopic soft tissue calcification and ossification. Recent reports have pointed out similarities between pseudohypoparathyroidism and myositis ossificans progressiva.[7,11]

Recently MacGregor and Whitehead[13] reviewed critically the reported cases of pseudohypoparathyroidism and concluded that chronic tetany with brachydactylia and/or ectopic calcification or bone formation is indisputable proof of the disease.

3. *Pseudo-pseudohypoparathyroidism.* None of the 7 cases of this entity reported in the literature has had intracranial calcification. Case III of this report represents an apparently spontaneous conversion from pseudohypoparathyroidism to pseudo-pseudohypoparathyroidism, and the patient now shows calcification in the basal ganglia of the brain. It would be premature to make any generalizations from this single case, but the patient does illustrate an intimate relationship between these two entities. The fact that the patient's mother also has pseudo-pseudohypoparathyroidism makes it almost certain that these two diseases result from similar or identical genetic abnormalities. Another important possibility, pointed out to the authors by Professor C. E. Dent[8] and illustrated by Case III, is that some cases of pseudohypoparathyroidism may, with increasing age, acquire normal plasma levels of calcium and phosphorus. In this way they would then show the clinical and biochemical picture hitherto described as being due to a separate entity, for which the term pseudo-pseudohypoparathyroidism has up to now been used.

4. *Familial basal ganglia calcification.* It is known that hypoparathyroidism, pseudohypoparathyroidism, and pseudo-pseudohypoparathyroidism occur in families,[5,27,31,33] and familial occurrence of pseudohypoparathyroidism with basal ganglia calcification has been reported.[14]

Cases IV, V and VI in the present report fall into the group of patients with familial basal ganglia calcification who show no biochemical evidence of hypoparathyroidism on routine determinations of blood calcium and phosphorus. Case IV, however, was extensively investigated with balance studies in addition to routine blood calcium and phosphorus determinations, and the result of these investigations of the patient and also of an affected niece showed the presence of latent abnormalities in phosphorus metabolism.

It is tempting to include this group of patients with "idiopathic" familial basal ganglia calcification within the larger group of patients showing overt clinical and biochemical evidences of parathyroid hypofunction. However, until more extensive studies, beyond routine determinations of blood calcium and phosphorus, are done on families with such intracranial calcification, no meaningful conclusions regarding latent parathyroid dysfunction as the etiologic factor in this group can be made.

5. *Unclassified group.* The etiogenic factor(s) responsible for the intracranial calcification in the 3 patients included in this group remains obscure. Each showed features common to the other 2 and to reported cases of basal ganglia calcification in patients described in the four groups listed above.

ROENTGEN FINDINGS

When the calcification in the basal ganglia is extensive, little difficulty is encountered in the proper anatomic localization of the calcification, provided roentgenograms are obtained in the usual complement of views of the skull. Calcification in the dentate nuclei is commonly obscured in the lateral view by the mastoid bones, but is well demonstrated in the Towne's projection.

There do not appear to be any distinctive features in the pattern or extent of intracranial calcification which might serve to distinguish the groups of patients with basal ganglia calcification. The pattern and degree vary considerably from small, irregular, discrete shadows of increased density to large masses of coalescing calcification. The radiating pattern noted in some patients appears to be due to calcium deposition in the gray matter between the bundles of white fibers of the internal capsule.

There are a few difficulties in differential diagnosis that seem worth mentioning. Symmetric calcification in the choroid plexuses may cause some confusion. Usually, however, such calcification occurs in the region of the genu of the lateral ventricles, well posterior to the anatomic position of the basal ganglia.

Raised intracranial pressure with papilledema occurs in some patients with hypoparathyroidism and basal ganglia calcification, apparently due to cerebral edema. The bilateral symmetric character of the calcification should help to eliminate the possibility of a calcium-containing new growth. Case V of this report represents an unusual instance where the diagnosis of a space-occupying intracranial lesion was made on the basis of displacement of calcified basal ganglia.

Finally, calcific deposits in the basal ganglia due to tuberous sclerosis and toxoplasmosis have been seen at the The National Hospital. However, none of these has shown the dense, symmetric, bilateral calcification seen in the groups of patients discussed in this report. The clinical impression of toxoplasmosis in Case VII was never confirmed, and, in retrospect, the dense calcification seen in the basal ganglia of this patient would appear to be due to some other cause.

SUMMARY

A number of cases with calcification in the basal ganglia of the brain, visible on roentgenograms of the skull, has been presented, and the major disease entities in which such calcification occurs have been discussed. The roentgen appearance of this intracranial calcification and the points of differential diagnosis have been reviewed.

An instance of spontaneous conversion of pseudohypoparathyroidism into pseudo-pseudohypoparathyroidism, with the concomitant development of calcification in the basal ganglia, has also been presented. The suggestion is made that pseudohypothyroidism and the so-called pseudo-pseudohypoparathyroidism result from similar or identical genetic abnormalities, and that some cases of pseudohypoparathyroidism may, with increasing age, acquire normal plasma levels of calcium and phosphorus, in effect converting from one to the other disease.

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ROENTGENOLOGIC STUDY OF THE VASCULAR MARKINGS OF THE SKULL*

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THE most frequent and important roentgenographic examination of the skull and its contents in everyday radiologic practice is the direct study in various positions without special contrast media. This is true in spite of the great advance in the use of contrast studies. The correct analysis of a "plain" skull examination will often reward one immediately with the correct diagnosis.

The purpose of this paper is to review the known information about vascular markings in the skull and to present 2 cases with interesting observations.

The vascular markings in the skull as seen on roentgenograms can be separated anatomically into two categories: (1) markings due to the diploic vessels; and (2) markings of branches of meningeal arteries and veins. The diagnostic significance of these markings has been questioned by some writers. Changes in the diploic channels have been described in such conditions as hypertension, heart failure, syphilis, chronic sinusitis, etc. Because of this, the reliability and the significance of these roentgenographic findings have been doubted. Sosman (1935) stated that in 25 per cent of the cases of tumor of the skull he could predict the histologic diagnosis from the study of the plain skull roentgenogram.

The brain and overlying structures have three vascular systems: (1) cerebral and cerebellar; (2) meningeal-osseous; and (3) superficial system for soft parts and bone. The first system is not visualized in the plain roentgenograms of the skull. The most important vessels of the meningeal-osseous system are the middle meningeal arteries and veins and the diploic veins. The third system consists of subcutaneous arteries and veins and vessels of muscles and the periosteum. An exchange of vessels takes place between the second and third systems at the emissary foramina, and numerous communications are found among the different vessel systems as well as within each system. Most of these communications are of no significance under normal conditions, and, since in large part they lie wholly within the soft tissues and are not visible roentgenographically, they appear to be unimportant. However, knowledge of their existence is of great help in explaining many of the changes which may take place pathologically. Among these are the widened osseous veins in cases of meningiomas which do not involve the bone and widening of the meningeal grooves in arteriovenous malformations of the cerebral vessels.

THE MIDDLE MENINGEAL ARTERY

The middle meningeal artery, a branch of the internal maxillary artery, is one of the most important arteries of the cranium. Its channel is the most consistently visualized on skull roentgenograms and detail study of it should always be undertaken.

It enters the skull through the foramen spinosum. The size of this foramen is an indication as to the size of the artery. Unilateral widening of the foramen is usually present in cases of pathologic enlargement of the middle meningeal artery. A difference of more than 2 mm. in diameter between the foramina should be considered as being significant. The average size of the foramen is 2 mm. (minimum 1 mm., maximum 3.5 mm.). A difference of 1 mm. occurs in 2 per cent of the cases and of 1.5 mm. in 1 per cent. The minute nervus spinosus of

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Luschka and a small vein are also found in the foramen spinosum but are of no consequence regarding the size of the foramen.

In evaluating the size of the foramen, rare conditions, such as defects of the wall and duplication of the foramen or fusion with the foramen ovale as well as the possibility of the uncommon congenital absence of the foramen (0.4 per cent), should be considered.

The middle meningeal artery after entering the cranium runs forward in a groove on the greater wing of the sphenoid bone and divides into two branches: (a) The anterior branch, the larger, crosses the greater wing of the sphenoid angle of the parietal bone. It then divides into branches which spread out between the dura mater and the internal surface of the cranium, some upward to the vertex and some backward to the occipital region. (b) The posterior branch curves posteriorly on the squama of the temporal bone. On reaching the parietal bone, it divides into branches which supply the posterior part of the dura mater and the cranium. Thus the two branches of the middle meningeal artery are distributed partly to the dura mater but chiefly to the bone substance. They anastomose with the arteries of the opposite side, also with the anterior and posterior meningeal arteries.

The groove of the middle meningeal artery is discernible on roentgenograms after the age of two years. Beyond this age, bone is deposited progressively on the inner table of the skull, and the groove is deepened, becoming more distinct. Occasionally, complete cannalization of the groove occurs, and roentgenographically it has the appearance of a diploic canal. The groove is shared by both the artery and the vein. Therefore, the size of the groove does not correspond to the size of the artery, and differential diagnosis between the vein and artery is very difficult. In the groove of the posterior trunk and the posteriorly directed branches of the anterior trunk, the artery takes up the larger part of the groove. These portions of the groove have more regular outlines and progressively diminish in size; usually they dichotomize. Then they take a less regular course and become venous grooves.

The anterior main trunk is often accompanied by a large vein lying in the same groove. At the pterion the artery is usually tortuous. A tortuous groove for the artery itself is sometimes possible to distinguish within the main groove. If the veins are small they follow the artery in a common tortuous groove instead.

Usually there are two meningeal veins in the groove accompanying the artery. They are small when compared with the artery. Below the edge of the lesser wing of the sphenoid, occasionally, there is a small branch of the middle meningeal artery accompanied by veins. Distally, toward the bregma, the artery is usually straight and has no separate groove. Posteriorly, where the posterior branch crosses the temporoparietal suture, some tortuosity of the groove may be seen. A distinct arterial groove at the pterion may have a width of 1.5 mm. (maximum 2 mm.). Any marked difference in the size of the grooves on the two sides should be considered as abnormal.

The medial portion of the anterior and middle cranial fossae and a great portion of the posterior cranial fossa are not supplied by the middle meningeal artery. These regions are supplied by a number of smaller arteries from the vertebral and occipital arteries and the anterior and posterior ethmoidal arteries. These are called anterior and posterior meningeal arteries.

In the parasagittal region the veins assume a funnel shape, causing lacunae and depressions due to pacchionian bodies, which suggest the nature of the vessels.

During the entire course, many communications with the diploic vessels may be evident.

THE SPHENOPARIETAL SINUS

The sphenoparietal sinus groove is posterior to the coronal suture and terminates in the cavernous sinus. It is straight and conspicuous but occasionally may cause

difficulty in identification and differential diagnosis.

THE LACUNAE OR VENOUS LAKES

The lacunae or venous lakes are caused by the enlargement of the meningeal veins in the parasagittal area and communicate with the cerebral veins. They are not present at birth but develop later. The lacunae show considerable variation and usually have no diagnostic pathologic significance. Their identification and the demonstration of the "feeding vessels" are important in order to differentiate them from pathologic changes, such as metastatic disease.

THE DIPLOIC VEINS

The diploic veins vary considerably in number, size and distribution. They occupy channels in the diploë of the cranial bone structure. They are large and exhibit at irregular intervals pouch-like dilatations; their walls are thin and formed of endothelium resting upon a layer of elastic tissue. As long as the cranial bones are separable one from the other, the diploic markings are confined to particular bones but, when the sutures are obliterated, they unite with each other and increase in size becoming, occasionally, very prominent in older individuals.

The size and number of the canals are not related to the thickness of the bone or the consistency of the diploë.

Usually, four groups of diploic veins are distinguishable: (a) the frontal, which opens into the supraorbital vein and the superior sagittal sinus; (b) the anterior temporal, which is confined chiefly to the frontal bone and opens into the sphenoparietal sinus and one of the deep temporal veins; (c) the posterior temporal, which is confined to the parietal bone and ends in the transverse sinus; and (d) the occipital, which is the largest and is confined to the occipital bone. It empties into the occipital vein or the transverse sinus.

There are no similar veins in the squamous portion of the temporal bone because it is devoid of diploë.

The diploic veins are considered as nutrient veins of the calvarium, but they are important in the study of intracranial circulation. They may widen more quickly than other blood pathways since the thin lamellae of the diploë produce less resistance to the venous walls. The diploic veins communicate with the meningeal veins, lacunae, veins of the pericranium, venous sinuses and cerebral veins and may reflect any change in circulatory dynamics. They begin to appear after the third year of life. One should keep in mind that under pathologic conditions the diploic veins may behave as vicarious pathways. Such vascular pathways however, should not be considered as widened when this change is observed only locally. These diploic grooves should be followed from their point of origin to the point where they can be seen either to leave the skull or to empty into another vessel.

Cranial blood vessels, in general, should not be regarded as isolated vessels, but their communications with other vessels should be taken into account, whether these are visible on the roentgenograms or not.

THE EMISSARY VEINS

Many of the canals and foramina which connect the cranial cavity with the exterior soft parts contain veins which are called emissary. They are usually small and the majority are without significance because the changes in size cannot be observed roentgenographically. The most important exception is the occipital emissary vein. At the occipital protuberance one to four fine openings are found. These canals communicate with the occipital diploic canals and are caused by the emissary veins. They continue through the bone to foramina above the external protuberance on the outer surface of the occipital bone. Their width usually does not exceed 2 mm.

DISCUSSION

The knowledge of the normal vascular markings of the calvarium and their variations is essential before attempting to

diagnose any pathologic changes. In this respect, the wide use of air, angiographic studies and the progress of neurosurgery and neuroradiology in recent years not only have enhanced the diagnostic possibilities but have afforded opportunity for re-evaluation of the plain skull roentgenograms.

The pathologic changes of the vascular markings can be divided into two groups:

(1) *Decrease of the size of the diploic markings* or their complete obliteration, which occurs occasionally when a tumor is peripheral and produces direct pressure on the vessels. The decreased diploic markings may be seen in deep-lying and slowly growing tumors as a manifestation of increased intracranial pressure; rarely, in hydrocephalus. This alteration may be local and coincide with pressure atrophy of the internal plate and diploë. Rarely, even the meningeal vein grooves may decrease in size.

(2) *Widening, enlargement and tortuosity of the diploic markings.* Widening of the occipital emissary vein with a large defect in the midline of the occipital bone has been observed in slowly growing tumors with increased intracranial pressure. It appears as a round or funnel-shaped defect in the external table of the calvarium in the region of the occipital protuberance. One must differentiate this widening from pacchionian depressions and defects due to erosion of bone by tumor. Also, in advanced age the bony defect of the normal emissary foramen may appear wider.

The grooves of the middle meningeal arteries, when widened, may be straight or tortuous but maintain their course and branching. The pressure of the enlarged artery produces a sharp outline of the edges of the groove. When the enlargement of the vessel is slight, it is very difficult to identify it. Comparison with the opposite side should be helpful. Normally, no appreciable difference in the width of the arterial groove on the two sides is found. However, it should be remembered that in midline lesions bilateral widening of the grooves is usually observed because of the dual blood supply of the tumor.

The meningiomas, particularly of the parasagittal area, are notorious for the high incidence of widened grooves of the middle meningeal arteries. It is known that the blood supply of these tumors is dual. The external carotid artery, through the middle meningeal, supplies the center of the tumor. The internal carotid artery supplies the peripheral part of the tumor. Widened and often markedly tortuous grooves belonging to tributaries of the middle meningeal artery may be seen leading to the tumor area from this region. Large collecting venous channels are usually present extending into the adjacent areas. The increased volume of blood required for the growth of the tumor and supplied by the meningeal arteries is then manifested by prominent, wide and tortuous diploic veins, widely anastomosed with meningeal and cerebral vessels. After successful resection of a tumor, the prominent markings decrease and may return to normal in a period of several months or even years. When the disturbance of the circulation presumably remains, the restitution to normal is only partial. In posterior parasagittal meningiomas, occasionally, widening of the foramen transversarium of the second cervical vertebra is observed. This is caused by the vertebral artery which, through the meningeal branch, supplies the blood to the tumor, a phenomenon similar to the dilatation of the foramen spinosum. In such cases, the diameter of the foramen transversarium may reach 7–9 mm.

Apparently, the developing of the vascular channel changes requires a long time, probably about two years. These changes appear to be less dependent upon the rapidity of growth of the tumor itself than upon the disturbance in the circulation of the blood which follows the development of the tumor.

Prominent vascular markings are also observed in meningiomas of the convexity, wings of the sphenoid and olfactory groove. In olfactory groove meningiomas, occasionally we may see widening of the foramen opticum due to dilatation of the ophthalmic

artery. Tumors other than meningiomas, most frequently gliomas, may produce similar vascular changes but usually these are not as prominent. The evidence depends upon the vascularity of the tumor and the rate of growth.

Arteriovenous malformations or true hemangiomas may show vascular markings indistinguishable from those of meningiomas; usually, the prominent vascular markings have not only diagnostic value but also help to determine the location of the tumor. It should be remembered, however, that sometimes the tumor may be distant to the prominent vascular channels. In such instances these markings have no localizing significance.

Widening of the foramen spinosum is of great diagnostic value when it occurs. It assists in the evaluation of the vascular markings, particularly when the changes are slight, and in determining that the groove is arterial. When widening of the meningeal arterial grooves and of the ipsilateral foramen spinosum is demonstrated, one may almost certainly ascertain that one of two lesions is present: (a) convexity or parasagittal meningioma; or (b) arteriovenous aneurysm involving the middle meningeal and cerebral vessels. Differentiation between these two lesions is impossible from the vascular markings alone, but must be made on the basis of local bone changes and opaque studies.

Occasionally, in the local area of the tumor, several small prominent vascular markings which lead into large diploic channels are observed. It is possible that the diploic veins may abruptly arise as large markings, probably because direct communication between diploic and cerebral veins exists.

Widened diploic veins may be absent or may be present without arterial channel widening, apparently because the blood supply of the tumor is more cerebral than meningeal. When arterial groove changes are not present, the diagnostic value of wide diploic veins is minimal because of the wide range of normal variation.

It may be difficult on occasion to differentiate between an abnormally wide arterial channel and an insignificantly prominent diploic vein. In these cases advantage should be taken of the frequently forgotten anatomic fact that the squamous portion of the temporal bone has no diploë. Therefore, a prominent channel in the squamous area of the temporal bone of the skull should be considered as arterial, representing the posterior branch of the middle meningeal artery and thus enhancing the diagnostic implication. We have seen a few cases where this anatomic principle was not applied, and the diagnosis missed.

REPORT OF CASES

Case 1. This adult male was admitted to the hospital because of persistent headaches of five years' duration, Jacksonian seizures and difficulty in expressing himself. The neurologic examination revealed bilateral papilledema and right lower facial weakness.

Plain roentgenograms of the skull (Fig. 1) showed prominent vascular markings, mostly in the frontoparietal area, and just behind the dorsum of the sella turcica a dilated and tortu-

Fig. 1. Case 1. Left lateral view of the skull. Note the prominent vascular channels. A tortuous, wide groove is seen in the temporal area behind the dorsum sellae, and a second less prominent groove is seen just above the sella. This is a pathologically dilated meningeal groove because no diploic channels are normally present in this area.

FIG. 2. Case I. Posteroanterior view of the skull. Note that the prominent vascular channels are only on the left side, which is a significant and pathologic finding.

ous vascular groove, which extended posteriorly and merged with the other channels, was noted. On the basis that there is no diploë in this area of the temporal bone, this groove should have been attributed to a dilated posterior branch of the middle meningeal artery and thus considered as pathologic; however, this was overlooked, and the skull was considered as negative. A second prominent groove was seen just above the sella turcica. A posteroanterior roentgenogram of the skull (Fig. 2) demonstrated that the prominent vascular channels were unilateral, in the left temporoparietal area. This is an additional pathologic finding. Because of the abnormal neurologic findings, left carotid angiography was performed.

Figure 3 shows the abnormal small vessels with displacement of the middle cerebral artery and its branches, findings which are characteristic of meningioma. The slight widening of the loop of the pericallosal artery suggests moderately increased intracranial pressure with dilatation of the lateral ventricle.

A relatively large meningioma was successfully removed at surgery.

The external carotid artery was not injected;

FIG. 3. Case I. Left lateral view of the internal carotid angiogram, late arterial phase. Note the multiple tiny tumor vessels with displacement of the middle cerebral artery downwards and stretching of the anterior and posterior branches of the middle cerebral artery. Note the slight widening of the loop of the pericallosal artery. The findings are characteristic of meningioma.

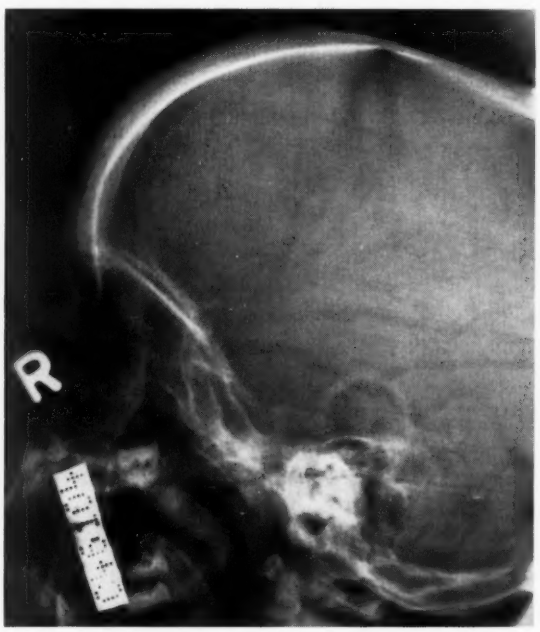

FIG. 4. Case II. Right lateral view of the skull. Note the very prominent, wide vascular grooves in the temporal region.

Fig. 5. Case II. Carotid angiogram. Right lateral view. Note the wide meningeal branches which correspond to the prominent vascular grooves seen in the plain roentgenogram (Fig. 4). Incomplete visualization of the internal carotid artery.

Fig. 6. Case II. Posteroanterior view of carotid angiogram. Note the abnormally wide arteries. Compare with Figure 5.

therefore, the dilated branches of the middle meningeal artery, observed in the plain roentgenograms of the skull, were not visualized. However, we do not believe there is any doubt about their existence and significance, particularly after the surgical findings.

The patient is well five years after the operation.

Case II. This two year old boy was admitted to another hospital, complaining of vague headaches.

A plain roentgenogram of the skull in the right lateral view (Fig. 4) showed very prominent vascular channels in the temporal area. The diploic vessels were not yet developed. Again, no particular significance was attributed to this finding. After the injection of the common carotid artery, the dilated branches of the middle meningeal artery were visualized (Fig. 5 and 6).

An angiomatous malformation was found at surgery.

SUMMARY

The vascular markings of the skull have been described. Their anatomic variation and the pathologic alterations have been discussed. The diagnostic possibilities on detail study of the plain roentgenograms of the skull have been emphasized.

Particular attention has been focused on the squamous portion of the temporal bone, where the absence of diploë and diploic channels is frequently not considered and possible diagnoses from the plain roentgenograms are overlooked.

Two interesting and unusual cases are reported.

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We are indebted to Doctor Oscar Sugar for the information on both cases and for providing the roentgenograms of the second case.

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ROENTGENOLOGICALLY CONTROLLED PLACEMENT OF THE NEEDLE IN THE TRIGEMINAL ROOT FOR THE TREATMENT OF TIC DOULOUREUX*

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TIC douloureux is a type of paroxysmal facial pain. It occurs usually in patients over forty years of age. There is a trigger zone, the lightest touch of which precipitates the pain. The trigger zone is usually in the skin or mucous membrane served by the second and third divisions of the trigeminal nerve.

Although the cause of the condition is rarely known, the production of analgesia of the trigger area usually prevents further attacks. Transitory numbness is produced by alcoholic injection or surgical avulsion of the peripheral branches of the nerve. Permanent numbness is produced by partial or complete destruction (chemical or surgical) of the posterior (sensory) root.[4–7,12]

Penman[8,10] described a method of alcoholic injection of the posterior root of the gasserian ganglion under roentgenologic control. The needle is inserted through the skin of the cheek at a point selected by preliminary roentgenograms. It penetrates the center of the foramen ovale and is then advanced in a plane parallel to the anterior surface of the petrous bone. It is planned that such a needle pierce the mandibular nerve at the foramen and pass through the gasserian ganglion into its posterior root. Its position in the foramen and its depth of penetration are confirmed by additional roentgenograms. After reviewing the previous roentgenologic methods of determining the position of the needle, Penman[8] concluded that the necessary information could best be obtained from two views—a postero-anterior oblique projection just off the axis of the needle and a submento-vertical projection.

ANATOMY

Neural Structures. The major neural structure in Meckel's cavity is the *pars triangularis*, that portion of the posterior root which widens and flattens to join the crescent-shaped gasserian ganglion (Fig. 1 and 2). This so-called triangular part (actually rhomboidal) is about 4 mm. long (along the fibers) and 3 mm. thick. It is a more or less densely matted meshwork bathed in spinal fluid.[2] The ganglion itself is 2 to 4 mm. long and 2 mm. thick. The intracranial length of the mandibular division is generally 5 or 6 mm.,[3] but may be shorter.[2] Hence, the tip of a needle inserted 10 to 15 mm. through the center of the foramen ovale and directed toward the center of the posterior root (as it crosses the petrous ridge) may come to lie within the triangular part of the posterior root. The fibers of the posterior (sensory) root are more or less separable into three groups as they approach the ganglion. The third division lies inferolaterally, the second division, intermediate in position, and the first division, superomedially. As a needle passes through the foramen and engages the mandibular nerve, at a depth of about 6 mm., its tip is ordinarily in the ganglion itself. Penman has shown that best results are often obtained when the needle tip is 10 to 15 mm. deep to the posterior edge of the foramen ovale as shown in the submento-vertical roentgenogram.

Bone Structures. When sighting through the cheek and foramen ovale of a skull (with calvarium removed), one can rotate the skull so that one sees the petrous ridge just above the inferior margin of the foramen (Fig. 3). That portion of the petrous ridge crossed by the posterior trigeminal root (*pars compacta*) is usually a clearly defined groove, the *incisura nervi trigemini*, the lateral margin of which is more prominent than the medial margin. The skull is

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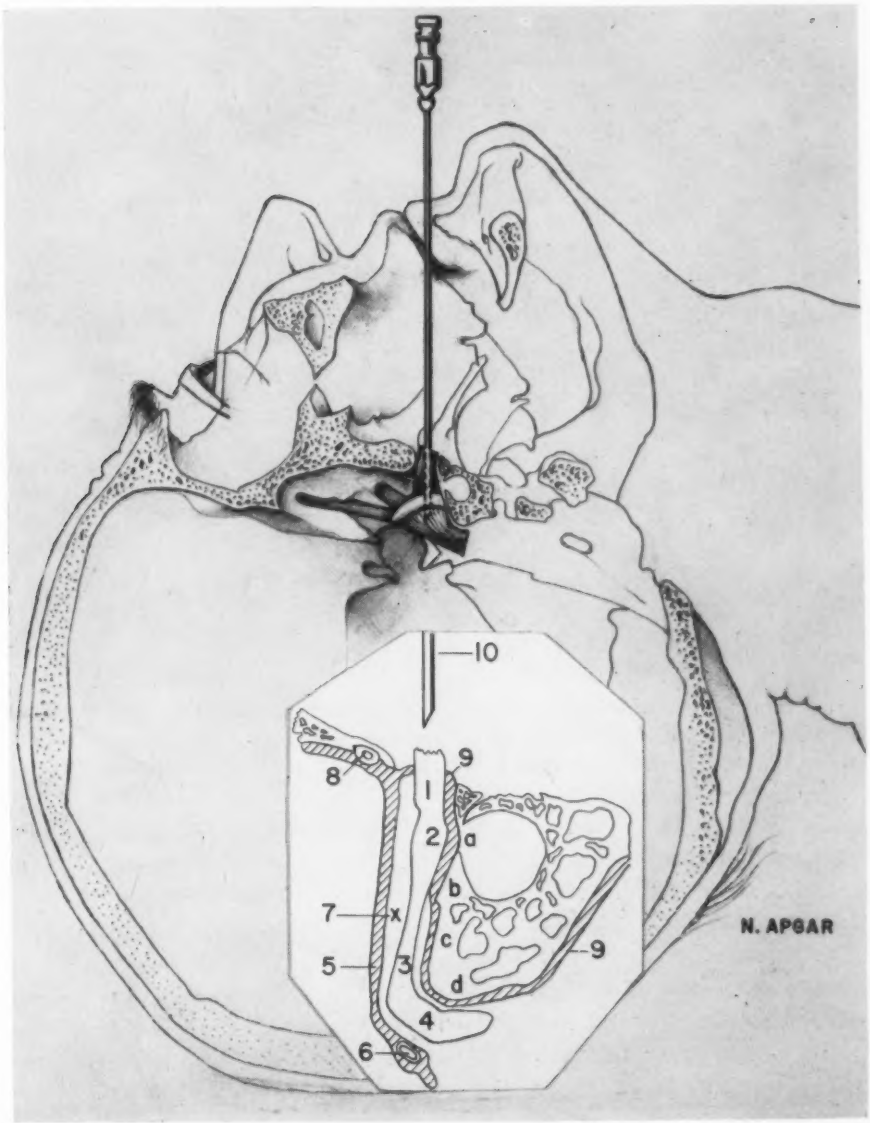

Fig. 1. Section of skull in a vertical plane rotated 20° away from the parasagittal. The correctly placed needle lies in this plane. The gasserian structures have been left intact. With the head in this position, the ophthalmic fibers lie at the lowermost level of Meckel's cavity. *Inset:* Enlargement of petrous portion with the section passing through the gasserian structures. I. Bone markings on anterior surface of petrous pyramid: (a) bone hiatus (the letter "a" lies in the carotid canal); (b) impression of gasserian ganglion; (c) impression of triangular plexus; (d) groove for posterior root. II. Soft tissues: 1, mandibular division; 2, gasserian ganglion; 3, triangular part of posterior root; 4, compact part of posterior root; 5, dural roof of Meckel's cavity, lying anterosuperior to the gasserian ganglion and the triangular part of the posterior root; 6, superior petrosal sinus; 7, cerebrospinal fluid in Meckel's cavity lying anterosuperior to the gasserian ganglion and the triangular part of the posterior root; 8, an intradural vein; 9, dura on the floor of Meckel's cavity, adherent to the ganglion and extending over the base of the skull; 10, needle approaching foramen ovale. (Reproduced by permission from *Neurology*.[1])

rotated until the center of this groove in the petrous ridge is in line with the center of the foramen ovale. The line of sight between the eye and a point 2 mm. above the incisura is the line of the needle insertion (Härtel's trigeminal axis).[4] The preliminary roentgenographic study determines the entry point and the head position which

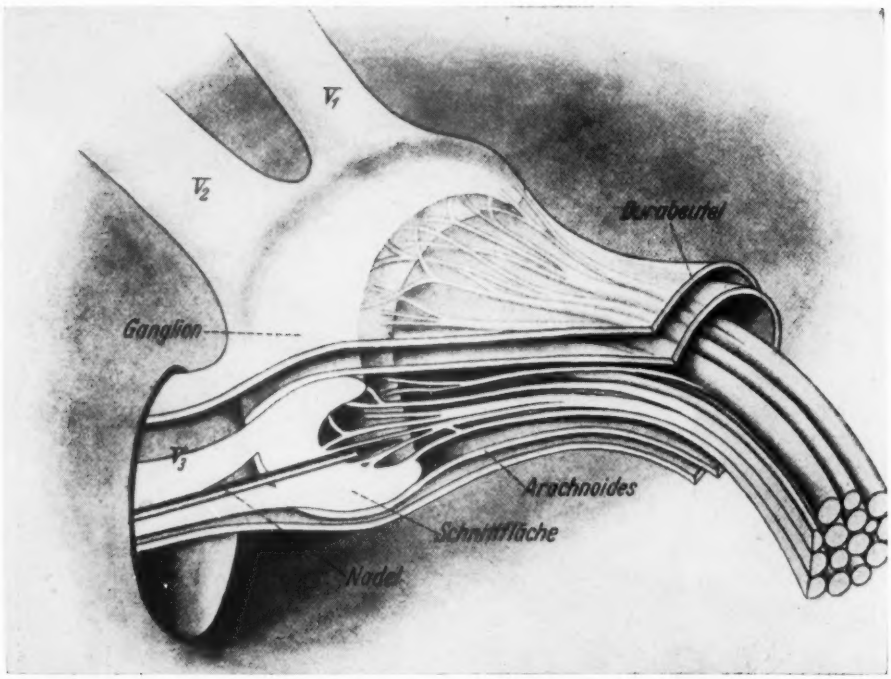

FIG. 2. Trigeminal root and ganglion (after Ferner). This sketch demonstrates that the trigeminal root is sometimes a loose meshwork of fibers bathed in spinal fluid and that the ganglion is actually "canoe-shaped" with walls 2 mm. thick. (Reproduced by permission from *Der Nervenarzt*.[2])

makes this line vertical (Fig. 3, 4 and 5). The head position can now be described by two angles—the bearing angle, between the vertical and the mid-sagittal plane, and the elevation angle, between the vertical and Reid's (orbito-meatal) baseline (Fig. 5). In 50 dried skulls, the range of the elevation angle was 55°–75° with an average of 65°; the range of the bearing angle was 8°–22°, an average of 15°.

MATERIALS

The purpose of this paper is to describe a technique for roentgenologically controlling the placement of the needle in the trigeminal root for the purpose of treating tic douloureux. For proper performance the following materials are needed:

1. A 2 cm. square wire grid (see text).
2. Two "Angligners" (Fig. 5) (protractors with long stylus manufactured by General Electric Company.)
3. Four transparent vertical guides (Fig. 5).
4. Syringes: (a) Two tuberculin syringes, each 1 cc. graduated in hundredths of a cubic centimeter; (b) a 5 cc. syringe for subcutaneous injection of procaine.
5. Needles: (a) Two needles—21-gauge (0.8 mm. external diameter) 4 inches (10 cm.) long with trochar-pointed stylets. This point avoids deflection in tissues as the needle is introduced; (b) one needle—22-gauge, 3 inches long, to inject local anesthetic.
6. Penman 1 cm. stop for the needle.
7. Dried skull with calvarium removed.
8. Ampules of absolute alcohol (each 2 cc.).
9. Ampules of procaine 2 per cent (each 2 cc.) or 1 bottle of 30 cc. of xylocaine 1 per cent.
10. Sterile gauze squares, 3×3 inches.
11. Sharp pencil, soft lead.
12. Gentian violet.
13. Prepared cotton swabs (applicators).
14. Transparent ruler.
15. Antiseptic (*e.g.*, 70 per cent alcohol).

PRELIMINARY ROENTGENOGRAPHY

In order to determine the point of entry of the needle, preliminary roentgenograms are taken in the following manner:

1. A mark is placed on the cheek with gentian violet at the approximate entry point at the level of the corner of the mouth, midway between the coronoid process of the mandible and the lateral aspect of the maxillary sinus (Fig. 3*B*).

2. The center of the grid is placed on the approximate entry point and taped to the cheek with transparent tape. The grid used by the authors is modified from that of Penman and consists of wires 1 cm. apart, making up a square 2 cm. on each side (Fig. 3*B*).

3. With gentian violet the orbito-meatal line (from the external canthus of the eye to the external auditory meatus on the involved side) and the mid-sagittal line on the forehead and nose (Fig. 3*B*) are drawn.

4. With the roentgenographic table horizontal, the patient lies supine on a mattress beyond which his head can be extended (Fig. 6). The head is extended to an eleva-

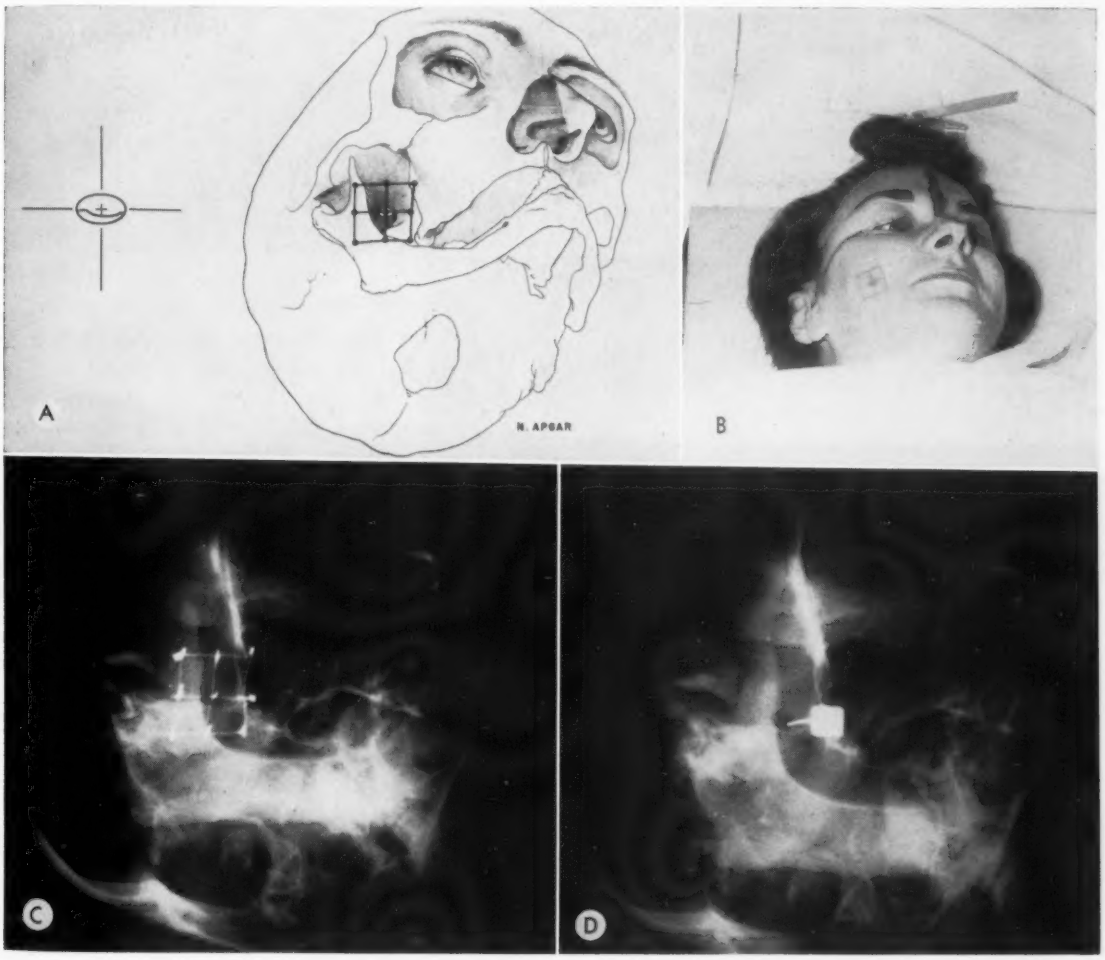

Fig. 3. (*A*) Sketch of foramen ovale and petrous ridge through right cheek from above with the skin grid in place. Insert at left demonstrates the *incisura nervi trigemini* as seen through the foramen ovale. (*B*) View from above with patient in position for preliminary roentgenogram illustrating mid-sagittal line, Reid's base line, and grid in place, centered over approximate entry point on right cheek. (*C*) Preliminary anterosuperior oblique roentgenogram—right side. Note foramen ovale, *incisura nervi trigemini* and grid. (*D*) Anteroposterior oblique roentgenogram with needle tip through foramen. (*C* and *D* reproduced by permission from *Neurology*.[1])

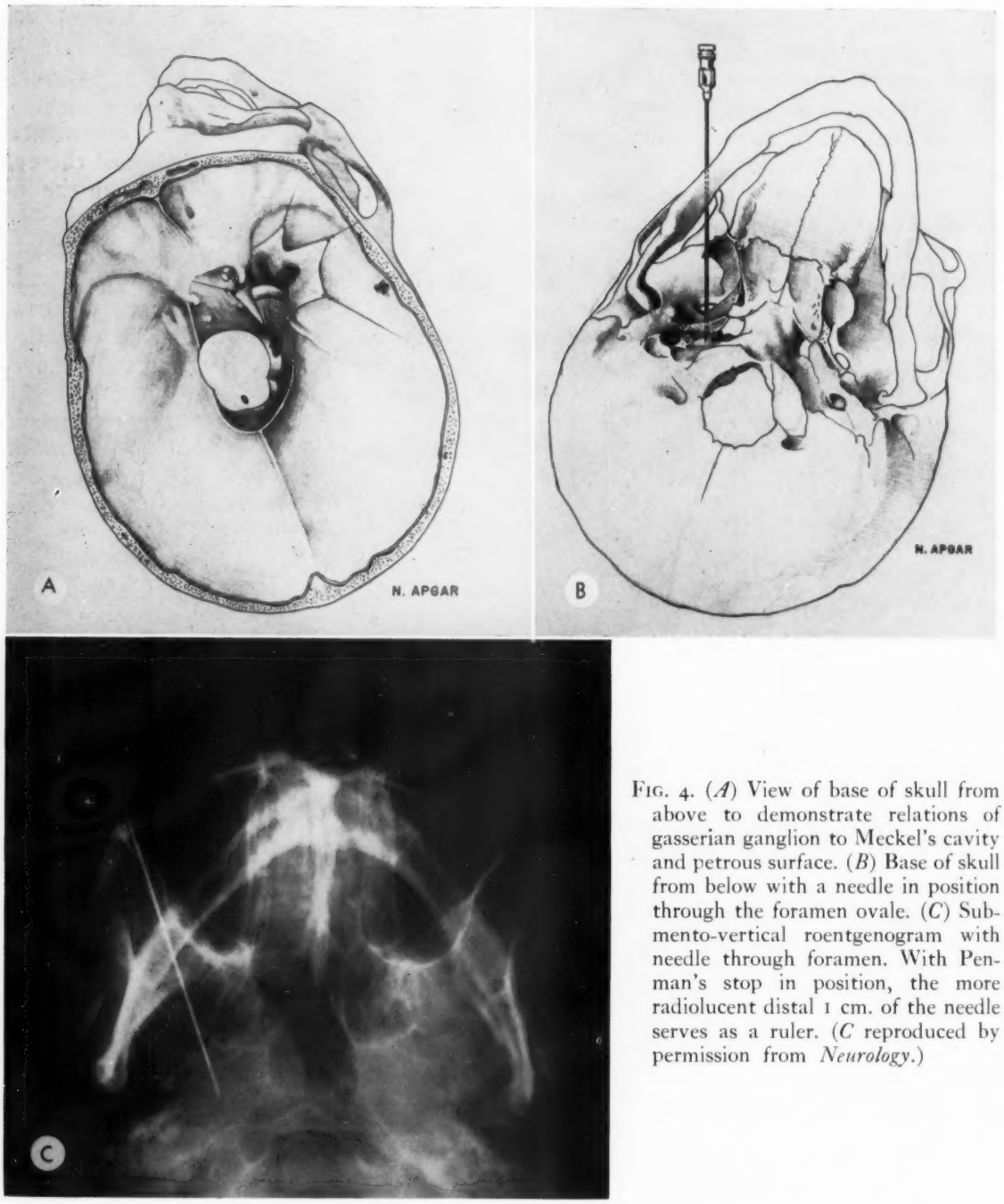

FIG. 4. (*A*) View of base of skull from above to demonstrate relations of gasserian ganglion to Meckel's cavity and petrous surface. (*B*) Base of skull from below with a needle in position through the foramen ovale. (*C*) Submento-vertical roentgenogram with needle through foramen. With Penman's stop in position, the more radiolucent distal 1 cm. of the needle serves as a ruler. (*C* reproduced by permission from *Neurology*.)

tion angle of 65°. It need not be brought into contact with the table, but can be supported by pieces of sponge rubber. The face is rotated away from the side of involvement until the bearing angle is 15° and positioned so that the approximate entry point is over the midline of the table. The elevation and bearing angles are accurately measured by the radiologist at the side of the table and the technician at the head of the table with the aid of "Angligners" (Fig. 5 and 6). It is important in sighting to be sure that the eye is at the level of the object being sighted. With a patient unable to co-

operate completely, it may be necessary to have the head held in place by another person. Adjustment of the elevation angle should be at right angles to the coronal plane; adjustment of the bearing angle, at right angles to the sagittal plane. With the aid of 2 vertical plastic guides, the central ray is directed through the approximate entry point (Fig. 5). Ten kilovolts are added to the usual factors so that it is possible to shorten the developing time to one minute.

5. Three roentgenograms are taken: the first, with an elevation angle of 65° and a bearing angle of 15°; the second, 68° and 18°; and the third, 70° and 20° (Fig. 3).

6. A preliminary roentgenogram in the submento-vertical view of the skull is taken (Fig. 4). This view can be omitted after some experience in studying wet films in this projection.

Interpretation of Preliminary Roentgenograms. The films are developed rapidly and studied at once. The optimum position of the head is that in which the center of the *incisura nervi trigemini* appears on the roentgenogram just above the center of the inferior margin of the foramen ovale (Fig. 3). With the head in this position, a needle inserted vertically downward will follow the path of the central roentgen ray. It will pass through the center of the foramen ovale parallel to the anterior petrous surface and toward the center of the pars triangularis (as it crosses the incisura of the petrous ridge).

In about half of the cases, one of the preliminary roentgenograms shows this optimum relationship. If necessary, additional roentgenograms are made with a slight change of the elevation angle or bearing angle or both. The degree of adjustment is based on a comparative study of the three initial roentgenograms. As an aid in determining this, the dried skull can be held in the position of the best of the three views and rotated to the desired position while noting the degree of rotation.

When the optimum head position is obtained, the final entry point is determined from that portion of the grid which superimposes the center of the incisura nervi trigemini and the center of the foramen ovale. The final entry point is marked on the skin with a sharp, soft, graphite pencil.

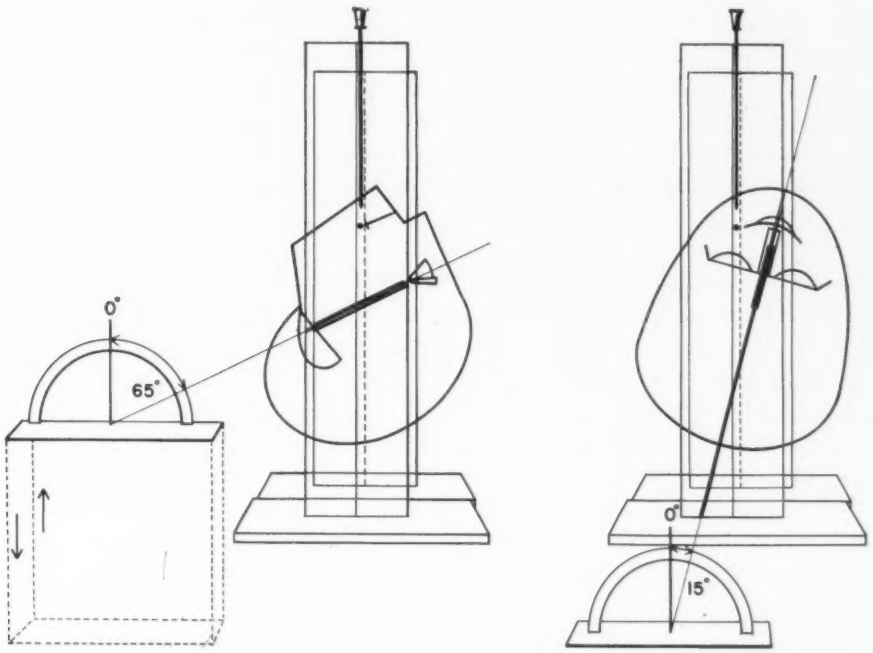

Fig. 5. Sketch of head, transparent vertical guides, "Angligner," and needle as observed from side and from head end of table.

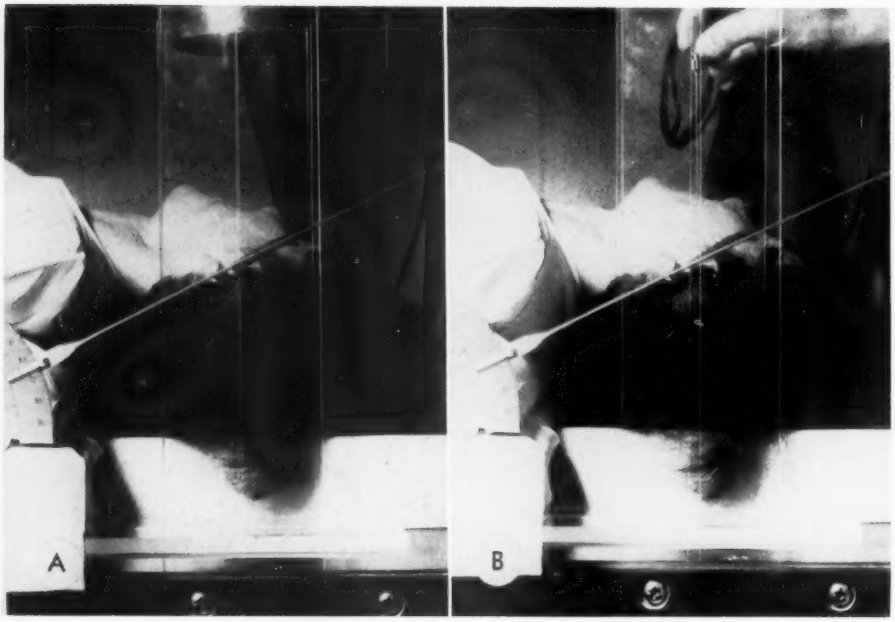

FIG. 6. Photographs of patient's head, vertical guides, and "Angligner" from side of table for preliminary roentgenography (*A*) and needle insertion (*B*).

NEEDLE INSERTION

Technique. General anesthesia is not used. The skin at the final entry point is chemically sterilized. With the aid of "Angligners," the skull is placed and maintained in the optimum position, as determined by the preliminary roentgenograms. To guide the needle, each observer places his transparent guides so that the centerline of one is 0.5 cm. to the right and that of the other 0.5 cm. to the left of the final entry point. Two per cent procaine or 1 per cent xylocaine is injected to a depth of 3 inches. A 21-gauge, 10 cm. long trocar-pointed needle is placed in a vertical position with its point touching the skin at the final entry point. As it is advanced slowly through the skin and the tissues of the cheek, its vertical direction is maintained carefully by comparing its direction in a biplane manner with the vertical transparent guides (Fig. 5). As the needle enters the foramen ovale, the patient experiences brief sharp pain in the mandibular division. The surgeon feels resistance and sudden yielding of the neural tissue. The needle is inserted approximately 12 mm. beyond this point of resistance. The stylet is withdrawn to see whether blood or spinal fluid escapes. Such fluid escape will be discussed later.

Roentgenograms for Determination of the Position of the Needle. If neither blood nor spinal fluid is obtained, two check roentgenograms are made. For the first roentgenogram, the central ray is directed through the needle. If the needle were truly vertical, the hub would conceal the point and the foramen on the roentgenograms. For this reason, the face is rotated 5° toward the unaffected side so that the hub of the needle will appear on the roentgenogram just medial to the needle tip and the foramen. This view is called the needle anteroposterior oblique view (Fig. 3). The second check roentgenogram, the needle submento-vertical view, is made in the usual manner except that the tube is shifted 2 cm. lateral to the midline (toward the affected side) and 2 cm. anterior to the external auditory canal. As a result, the central ray is directed through the foramen ovale. This view is made with the stylet withdrawn 1 cm. and held there by the stop as suggested by Penman. With the tip of the stylet 1 cm. from the tip of the needle, the distal 1 cm. of the needle is less dense in

the film. This provides an accurate method of measuring intracranial penetration (Fig. 4). If the trochar-pointed stylet protrudes beyond the needle, allowance is made.

Adjustment of the Depth of the Needle If It Has Entered the Foramen Ovale. If the roentgenograms show that the needle has been correctly placed, its depth can be adjusted by pressing a small transparent ruler against the skin alongside and parallel to the needle and measuring the distance that the hub moves as the needle is withdrawn or inserted further. The amount of adjustment is determined by a study of the submento-vertical view with the Penman 1 cm. stop. After the adjustment is made, it is wise to confirm the new position by an additional submento-vertical roentgenogram. To help prevent inadvertent movement of the needle during injection, a needle holder is attached to the surface of the needle at the skin level. If third division anesthesia only is desired, the tip of the needle is inserted to an approximate depth of 10–12 mm. beyond the posterior margin of the foramen ovale; for the second and third divisions, 12–14 mm.; for the second division alone, 14 mm. These considerations apply if no spinal fluid emerges from the needle, thus implying that the posterior root is a relatively dense meshwork.[2]

Test Injection of Procaine. The control roentgenograms have given the relationship of the needle tip to the base of the skull, especially the foramen ovale. To test the relation of the tip of the needle to the neural tissue, 0.03 cc. of 2 per cent procaine or 1 per cent xylocaine is injected. Both touch and pain sensations are tested in several areas of the face and recorded. From the roentgenograms and these sensory data, we judge the exact site of the needle tip in relation to the posterior root. We consider also that procaine diffuses more widely than absolute alcohol and that there are individual variations in the length of the posterior root and ganglion. When necessary, we make further adjustments of the depth of the needle and take additional submento-vertical roentgenograms. For example, if first division effect is produced, the needle is withdrawn at least 2–3 mm. before another injection is made.

Alcohol Injection. If appropriate second and/or third division anesthesia is obtained by procaine, the head is placed so that the needle is vertical, thus bringing the first division fibers lowest. A single injection of 0.05 cc. of absolute alcohol is now made with a tuberculin syringe. If appropriate numbness is obtained (which will take place within four minutes, if at all), there are recorded the time of the injection and sensory responses in each of the three trigeminal divisions. As the immediate effect of the alcohol wears off (usually within twenty minutes), additional injections of alcohol are made as necessary—always with an interval of at least four minutes between injections. The time and results are recorded before each additional injection is made. We have come to accept Härtel's recommendation of a maximum of 0.3 cc. of absolute alcohol. We had to exceed this figure in only 4 of 61 cases. When sufficient numbness to suppress production of pain in the trigger area lasts at least thirty-five minutes, the needle is withdrawn and the patient is permitted to sit up and then leave. If there is any corneal anesthesia, the patient is warned against rubbing his eye.

Problems and Complications of Needle Insertion. If the needle fails to enter the foramen, it will strike the bone under-surface of the floor of the middle cranial fossa at a depth of about 5 cm. Then submento-vertical and needle anteroposterior oblique roentgenograms are made. In our earlier experience, when the needle failed to enter, we used the check roentgenogram to guide insertion of a second needle in relationship to the first. More recently, we have rechecked the position of the skull (bearing and elevation angles) and of the needle (vertical as checked in 2 directions). This has invariably indicated that the patient's head or skin had moved. We now withdraw the needle almost to the skin, re-position the head, and re-insert the needle. Because the deeper portion of the needle is fixed by

the tissues, only the slightest adjustment in the direction of the needle can be made without withdrawing. With careful preliminary roentgenograms, selection of the entry point and control of the position of the head and needle, it is usual to enter the foramen ovale on the first attempt.

If blood emerges when the stylet is withdrawn after a needle insertion, the needle is slowly withdrawn until blood no longer appears. The needle is cleared with sterile saline solution and the position of the needle checked roentgenographically. Occasionally, a branch of the internal maxillary artery is perforated and a hematoma develops in the cheek. This does not interfere with continuing the procedure. Rapidly dripping blood, in our experience, twice came from the middle meningeal artery at the foramen spinosum. Blood emerging under low pressure may have come from the venous plexus around Meckel's cavity. This has occurred a few times. Although it may be possible to strike the internal carotid artery in the petrous bone, we have not done so.

After insertion of the needle through the foramen ovale, cerebrospinal fluid may escape when the stylet is removed. Ferner's dissections show that the preganglionic fibers are actually a more or less loose meshwork of fibers bathed in spinal fluid. This indicates that a needle accurately placed in the preganglionic area (sensory root) might still draw spinal fluid. In this event, the needle is withdrawn slowly until the flow of fluid just stops. We consider that the needle tip is then barely within the ganglion itself. This is true, even if it is as close as 4 mm. to the posterior edge of the foramen ovale.[2] The patient's face is rotated 15° to the unaffected side, and the alcohol injected as previously described. Under these circumstances, if 0.05 cc. of alcohol is ineffective, one may inject 0.10 cc. (Fig. 2).

Occasionally, with the needle through the foramen and no cerebrospinal fluid flow, the desired result is not obtained with the test dose of procaine at various depths. This means that the needle tip is incorrectly placed. This is more likely to occur when the needle has passed through the foramen away from its center, or is not in the correct plane or line. In such cases, which are rare, it may be necessary to re-insert the needle.

In all but one of our cases, the patient was supine. The exceptional patient had asthma and could not tolerate lying down. Hence, we carried out the procedure with the roentgenographic table vertical and the patient sitting with the head hyperextended.

RESULTS

From October, 1954 through October, 1958, we have used this method in 60 successive patients with trigeminal neuralgia. One patient had bilateral trigeminal neuralgia; hence, there were 61 instances. Numbness was produced in all patients and pain completely relieved, initially. However, in 10 of the patients, the pain recurred and another injection was necessary. One patient of the 60 injected underwent surgical rhizotomy later. He was in a distant city at the time his pain recurred. In 8 patients, there was total corneal anesthesia. In 2, there were brief periods of superficial corneal abrasion, but in no case was there keratitis or the need for tarsorrhaphy. In 1, corneal sensation returned in one year. About half of the patients have had postinjection paresthesias. Of these, only 3 were severe. The minor complications and more details of our sensory testing and results are presented elsewhere.[1]

DISCUSSION

We believe this method to be effective and safe. It carries practically no risk of death and a very low risk of neuroparalytic keratitis.

While following Penman's general approach, we have used preliminary roentgenography not only to select the entry point, but also to determine the position of the head for the vertically downward insertion of the needle. After the completion of the preliminary roentgenographic study,

the duration of the procedure (insertion, check roentgenography, needle adjustment and testing) has averaged one hour. Once the needle has been placed in the correct position, the patient can be removed from the roentgenographic table so that further injecting and testing can be done in another room. This precise, sometimes difficult, work can be done only with patience and the cooperation of the patient, radiologist and surgeon.

SUMMARY

A method of inserting a needle into the posterior root of the gasserian ganglion is presented. A preliminary roentgenographic study determines the exact position of the head and the entry point of the needle. After the needle is inserted, further roentgenographic study indicates the relationship of the needle tip to the base of the skull and permits accurate adjustment of its depth. Then procaine (2 per cent, 0.03 cc.) is injected to determine the position of the needle within the posterior root. Finally, therapeutic injections of 0.05 cc. of absolute alcohol are made as indicated by careful sensory and motor testing. This procedure requires patience, cooperation and a precise knowledge of anatomy. In four years, we have found it safe and effective in relieving trigeminal neuralgia in 61 consecutive instances (60 patients). This paper emphasizes the roentgenologic technique.

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Transparent vertical guides were suggested and designed by Mr. Stanley Stevens, X-ray Technician, Onondaga General Hospital, Syracuse, New York. The trochar-pointed stylet and needle were designed and prepared by Mr. Werner Spingler, B. G. Sulzle Company, Syracuse, New York.

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ROENTGENOGRAPHIC ANALYSIS OF RENAL MASS LESIONS (CYSTS AND TUMORS)*

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IN THE adult patient the great majority of mass lesions of the kidney are either simple cysts or primary renal carcinomas (hypernephromas). While clinical and laboratory findings in these two entities are sometimes diagnostically helpful, the major burden of differentiation rests on the proper interpretation of roentgenograms. It has been repeatedly stated that such differentiation is impossible by roentgenographic means and that "infallible guides for a constantly correct diagnosis without surgical exploration"[5] are lacking. However, experience in recent years with renal angiography and nephrotomography in the study of renal mass lesions (cysts and tumors) has created the impression in our minds that a more critical analysis of the pyelographic findings of such lesions might permit more accurate diagnosis than previously thought possible. Such an optimistic attitude has been expressed by various authorities[1,2,4,6,7] and recently Shivers and Axilrod[8] stated that "careful consideration of all of the aspects of the clinical picture together with detailed consideration of pyelographic changes will lead to a correct pre-operative diagnosis in a surprisingly large percentage of cases."

MATERIAL

To test the possibilities and limitations of simple roentgen studies in the differentiation of renal cyst and tumor, we collected 24 examples of primary renal tumor (Fig. 1) and 18 examples of simple renal cysts (Fig. 2) encountered in the last eight years at Harper Hospital, Detroit, Michigan. All patients concerned had been subjected to retrograde pyelography and in each a surgical or postmortem tissue diagnosis had been obtained.

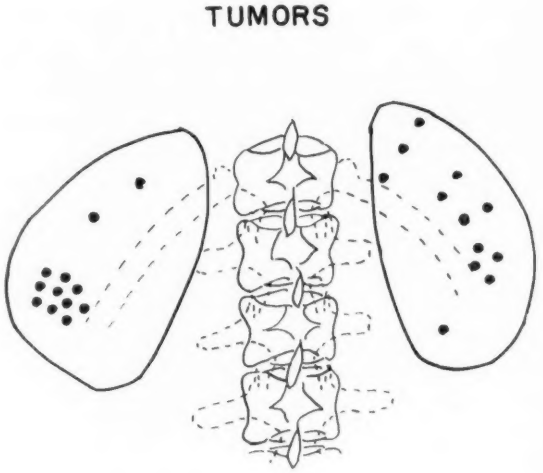

Fig. 1. Distribution of 24 renal tumors.

ROENTGEN FEATURES

Roentgenographically, the lesions presented as masses contiguous with or attached to the kidney, producing renal outline or pyelographic deformities of such nature as to evoke the question—cyst or tumor? These roentgen features were evaluated as follows:

Size of Kidney. Renal size can be evaluated in most instances by comparison of the abnormal kidney with the normal. In the majority of kidneys in which primary tumor was present, the renal outline itself was enlarged (22 of 24 cases); whereas, in renal cyst, actual increase in size of the kidney from which the cyst arose was uncommon (3 of 18 cases).

Size of Mass. Indistinct outlines of the mass characterized renal tumors, preventing in most instances any attempt at size determination. In fact, in only 7 of the tu-

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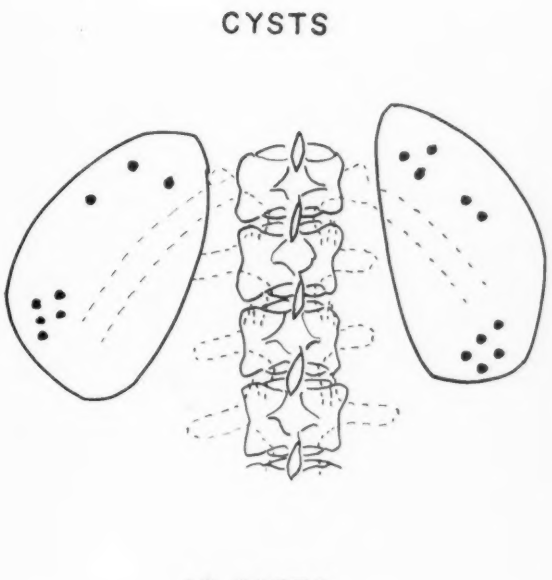

Fig. 2. Distribution of 18 renal cysts.

mor cases could the diameter of the mass be estimated by roentgenography. Sharp outlines were exhibited by renal cysts, particularly those arising from the lateral surface or poles of the kidney, and in these the diameters were easily determined. Average diameter of the renal cysts as measured on the roentgenograms was 10 cm.

The mass of renal tumor is contiguous with the kidney and has density of equal or greater degree than the surrounding kidney tissue. This is also true of most renal cysts, but a few demonstrated decreased density as compared to the underlying or overlying renal substance and produced a "double shadow," which is virtually characteristic of renal cyst. Four of our 18 examples of renal cyst exhibited such a "double shadow" (Fig. 3). This sign should be carefully sought, particularly in lower pole mass lesions. Laminagraphy may be of value in eliciting this sign. Of similar nature and significance is the observation, reiterated by Shivers and Axilrod,[8] that the psoas line can frequently be seen through the cyst; whereas, in solid tumors the psoas line may be obliterated (Fig. 4 and 5).

Calcification. Calcification is said to be roentgenographically discernible in about

Fig. 3. Cyst of lower pole of right kidney. Darker central area representing renal parenchyma; lighter peripheral area representing cyst, forming so-called "double contour."

Fig. 4. Large cyst of lower pole of left kidney. Psoas muscle clearly visible through overlying cyst.

15 per cent of renal carcinomas[3] and is rare in simple renal cysts. Tumor calcifications are usually centrally located and may be flocculent, linear, fleck-like, spiculated or amorphous. Linear or curvilinear calcific streaks are sometimes shown at the periphery of simple cysts. Four of our 24 renal carcinomas contained calcific areas. None of the cysts showed calcific inclusions.

Renal Pelvis. Depending upon the location of the mass, cyst or tumor within the kidney, the renal pelvis may be entirely uninvolved or may be displaced or compressed. Masses in the central or medial portion of the kidney are most likely to produce such pelvic deformity. The pelvic deformity is not characteristic of either cyst or tumor unless frank infiltration of the pelvic wall can be demonstrated, but even here caution must be exercised. In 3 of our renal tumors and in 2 of our renal cysts, the renal pelves were displaced and/or compressed, but in none of the 5 cases was the renal pelvic involvement alone of aid in differentiation (Fig. 6).

Calyceal Disturbance. Involvement of the major and minor renal calyces can be due to either renal cyst or tumor. In general, such calyceal involvement is one of four types or a combination of these types: (1) elongation, (2) displacement, (3) compression, and (4) amputation (obliteration). Each of these types of calyceal involvement was confirmed or denied in the pyelographic pattern of the tumor and cyst cases in our series. Of considerable aid in appreciation of the four defect types is comparison with the opposite or "normal" calyceal pattern.

Fig. 5. Tumor of lower pole of left kidney. Psoas muscle obliterated by tumor.

Fig. 6. Large central renal tumor obliterating renal pelvis.

1. *Elongation.* As a tumor or cystic mass within the kidney substance enlarges, the neighboring calyces are stretched in a centrifugal fashion (Fig. 7*A*). Such calyceal elongation appears to be more commonly associated with renal tumor, as in our series it was seen in 19 of 24 pyelograms, and various combinations of elongation with displacement, compression and amputation were commonly recorded (Table 1). Calyceal elongation in combination with displacement, compression or amputation was encountered in less than half as many cases of renal cyst. The combination of calyceal elongation and amputation or obliteration of the calyceal tip comprises a special configuration for renal tumor and was not visualized in any renal cyst in our series.

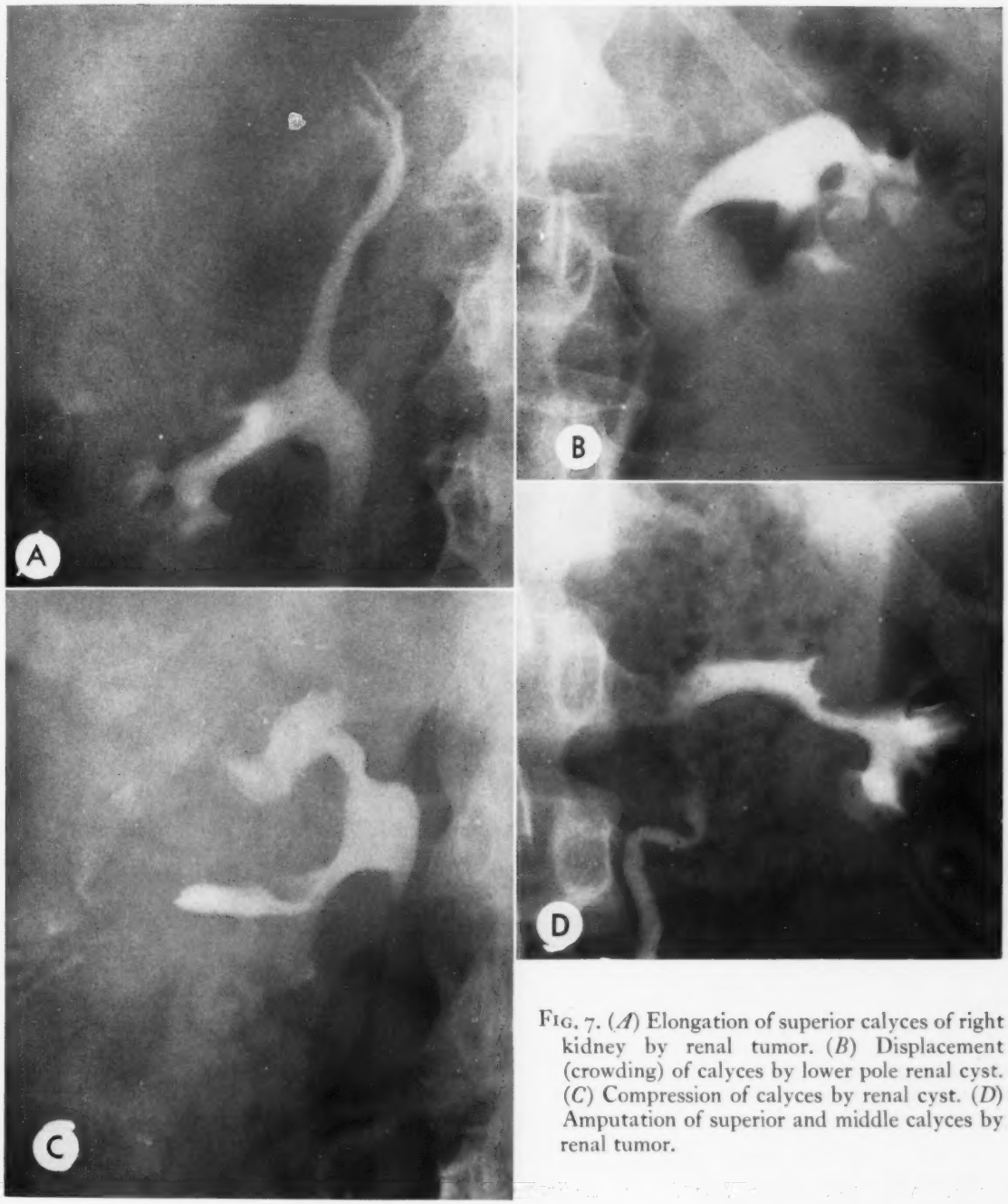

Fig. 7. (*A*) Elongation of superior calyces of right kidney by renal tumor. (*B*) Displacement (crowding) of calyces by lower pole renal cyst. (*C*) Compression of calyces by renal cyst. (*D*) Amputation of superior and middle calyces by renal tumor.

2. *Displacement.* An expanding renal mass can displace calyces toward each other or separate them (Fig. 7*B*). Separation of calyces is frequently seen in both renal cyst and tumor, particularly if the lesion is centrally located in the kidney. This type of calyceal displacement is most difficult to assess, unless combined with calyceal compression, because of the wide variance in spacing of the calyces in the normal pyelographic pattern. Comparison with the pyelocalyceal pattern of the unaffected kidney is the only recourse but is not universally decisive.

Displacement of calyces toward each other, or "crowding" of calyces is more

commonly an attribute of cyst, occurring in 5 of our 18 cases. Calyceal crowding is particularly prevalent with large lower pole cysts.

3. *Compression.* Calyceal compression is a natural result of expansion of a renal mass in juxtaposition to the calyceal system (Fig. 7*C*). The involved calyx or calyces appear, in the frontal pyelogram, to be widened. Occasionally, however, dilatation of the calyx is simulated by a compressed calyx and here the use of oblique and lateral pyelograms is particularly indicated to display the degree of calyceal compression. Compression was produced by almost an equal number of our cases exhibiting cyst or tumor and, therefore, in itself is of little differential diagnostic value.

4. *Amputation.* Amputation (obliteration) of a calyx appears as complete occlusion of a major or minor calyx at its origin or, more likely, as an irregularity of the calyceal tip (Fig. 7*D*). Although amputation can be mimicked by a blood clot filling the calyceal lumen or by inflammatory involvement of the mucosal lining of the calyx, in most instances actual infiltration of the calyx and pericalyceal tissue is responsible for the roentgenographic appearance of amputation. Therefore, true amputation is a very suggestive sign of renal neoplasm and appeared in one-half of our renal tumor cases and in only one of the renal cyst patients. In the latter example the surgical specimen revealed an infected simple renal cyst, the amputation of the renal calyx being produced by granulation tissue.

These various combinations of calyceal deformity—elongation, compression, displacement and amputation—are more frequently demonstrated in renal solid tumors than in renal cysts (Table I and II). The more complex or bizarre the pyelocalyceal deformity, the more likely the lesion is tumor than cyst. However, complete absence of calyceal involvement may be found with either renal tumor or cyst, as was the case in 3 renal tumors and 2 renal cysts of our series (Fig. 8 and 9).

TABLE I

CALYCEAL DISTURBANCE IN 24 RENAL TUMORS

Calyceal Disturbance	No. of Cases
Elongation	19
Displacement	12
Compression	13
Amputation	12
Elongation and Displacement	11
Elongation and Compression	10
Elongation and Amputation	11
Displacement and Compression	7
Displacement and Amputation	7
Compression and Amputation	5
All Four Disturbances	3
No Calyceal Disturbance	2

DISCUSSION

Careful analysis of renal size, contour and pyelocalyceal deformity yields a surprising amount of information about renal cysts and tumors. In a high percentage the correct diagnosis can be made by use of the criteria enumerated. Why then should not a definite and definitive diagnosis of cyst (benign) or tumor (malignant) be possible in all renal mass lesions? The reason is twofold. Primarily, there is a certain number of patients in whom the roentgenographic studies do not conclusively exhibit signs of

TABLE II

CALYCEAL DISTURBANCE IN 18 RENAL CYSTS

Calyceal Disturbance	No. of Cases
Elongation	7
Displacement	4
Compression	13
Amputation	1
Elongation and Displacement	3
Elongation and Compression	5
Elongation and Amputation	0
Displacement and Compression	4
Displacement and Amputation	0
Compression and Amputation	1
All Four Disturbances	0
No Calyceal Disturbance	3

either renal cyst or tumor (20 per cent of our 42 patients are in this group). Secondly, if renal cyst can be diagnosed by means of the roentgen findings, there is still the possibility that neoplasm coexists within the cyst, as is the case in about 3 per cent of simple renal cysts and 30 per cent of hemorrhagic cysts,[9] according to the literature. For these two compelling reasons alone, the didactic diagnosis of renal cyst or tumor is hazardous, to say the least. More complicated roentgenographic procedures, such as nephrotomography, renal angiography and renal cystography, can probably decrease the diagnostic error to a slight degree, but in the final analysis histologic confirmation in any case of renal mass lesion is the only satisfactory proof. These discouraging conclusions should not deter the roentgenologist from a sincere attempt at differentiation of renal cyst and tumor by the means at his disposal, and we believe that a maximum of information in this regard can be rendered by the careful analysis of pyelograms.

Fig. 8. Marked displacement of kidney by upper pole tumor but no significant pyelocalyceal deformity.

Fig. 9. Absence of significant pyelocalyceal deformity with large right renal cyst.

The simple roentgenographic features of renal cyst and tumor can be summarized as follows:

Renal Cyst

1. Kidney itself usually normal in size.
2. Mass attached to the periphery of the kidney in many cases; sharp outlines, permitting measurement of diameter of mass; may be less dense than kidney tissue and produce a "double shadow."
3. Calcification, if present, of linear or curvilinear type at periphery of cyst.
4. Renal pelvis may be displaced or compressed.
5. Calyceal elongation sometimes found, usually in combination with calyceal displacement or compression.
6. Calyceal displacement often takes the form of "crowding" together of calyces.
7. Calyceal compression common, particularly with centrally located cysts.
8. Calyceal amputation is rare.
9. Simple types of calyceal deformity.
10. May show no calyceal deformity, particularly if cyst is subcapsular.

Renal Tumor

1. Kidney enlarges with tumor.
2. Mass contiguous with body of kidney; indistinct outlines; mass often denser than kidney and may obliterate psoas line.

3. Calcification central or eccentric in location—punctate, linear or amorphous.
4. Renal pelvis may be displaced, compressed or invaded.
5. Calyceal elongation frequently found, often in combination with calyceal amputation.
6. Calyceal displacement often takes the form of separation of calyces.
7. Calyceal compression common, particularly with centrally located tumors.
8. Calyceal amputation is very common.
9. Complex or bizarre types of calyceal deformity.
10. May show no calyceal deformity if tumor is peripheral in location.

It appears advantageous to us to critically tabulate the various clinical and roentgenographic findings in the study of each case of renal mass lesion. For this purpose we employ a check form as illustrated in Table III.

TABLE III

Patient's Name		
H No.	Dr.	
A No.		
Age:	Sex:	
Symptoms:		
Pain		
Gross hematuria		
Other		
Signs:		
Palpable mass		
Hematuria		
Pyuria		
Cystoscopy:		
Hematuria		
Pyuria		
Function of kidney		
Other findings		
Pyelography:		
Plain film:		
Side of lesion	Right	Left
Size of kidney		
Visible mass		
Calcification		
Other findings		
Pyelogram:		
Pelvis		
Calyceal elongation		
Calyceal displacement		
Calyceal compression		
Calyceal amputation		
Other findings		

SUMMARY

By careful analysis of the roentgenographic features of renal mass lesions (cysts and tumors), it is possible to differentiate these entities in a high percentage of cases.

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THE FIVE MINUTE INTRAVENOUS PYELOGRAM AS A MEASURE OF RENAL FUNCTION*

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INTRAVENOUS urography was introduced in the United States in 1930 and has provided the members of the medical profession with a valuable method for investigation of lesions within the urinary system. The endeavor to broaden the scope of excretory pyelography from a method of identification of pathologic shadows to include a measure of renal function has been noted many times in the literature during the past twenty-five years.[2,5,8,10]

It is the purpose of this study to offer a simple method for the assessment of renal function based on the degree of opacification of the renal pelves in the five minute intravenous pyelogram as compared to the roentgenographic density of the ribs.

HISTORICAL REVIEW

The observation that excretory urography could be utilized to yield both anatomic and physiologic information about the dynamics of the urinary system was initially made by von Lichtenberg *et al.*[8,9] Braasch,[1] in 1931, noted that visualization of the contrast medium within the kidneys promptly after intravenous injection could be regarded as a positive indication of normal renal function. This principle was empirically demonstrated in a series of clinical and laboratory observations carried out by Smith and Leadbetter[6] in 1941, which correlated low phenolsulfonphthalein excretion levels in the urine with the inability of the kidneys to excrete urographic media in sufficient concentration to cast a roentgenographic shadow.

Several different techniques have been offered for the use of contrast media to determine renal function. In two independent series, Braasch and Emmett[2] and Strom and Cook[7] qualitatively observed that prompt appearance of the medium within the urinary bladder indicated good renal function. Quantitative measurement of renal function has been suggested by Findley *et al.*[3] by direct determination of the amount of iodine excreted in the urine after the intravenous injection of iodinated urographic media. Due to the utilization of cumbersome techniques or indefinite criteria, none of these procedures have found widespread application.

METHOD

In order to correlate renal function with the appearance of the intravenous pyelogram, 217 consecutive excretory urograms made at the Veterans Administration Hospital, Ann Arbor, Michigan, were reviewed. Preliminary preparation of patients undergoing intravenous pyelography uniformly consisted of a minimum of eight hours of food and fluid restriction. The radiopaque medium administered was 30 cc. of intravenous 50 per cent aqueous solution of 3,5-diacetamido-2,4,6-triiodobenzoate (hypaque). Renal function determinations were carried out using the Chapman and Halsted fifteen minute modification of the intravenous phenolsulfonphthalein test as introduced by Rowntree and Geraghty in 1910,[4] and the twenty-four hour creatinine clearance test.

RESULTS

A review of the intravenous urograms revealed 194 cases where the density of the five minute pyelogram exceeded the roentgenographic density of the ribs. Renal function data in this group were then studied yielding the results as shown in Figures 1 and 2. Uniformly, all showed a fifteen minute phenolsulfonphthalein of 15 per cent or greater with an over-all average of 29.9 per cent. Similarly, all showed a twenty-four hour creatinine clearance of greater than 75 liters with an over-all average of 133.5

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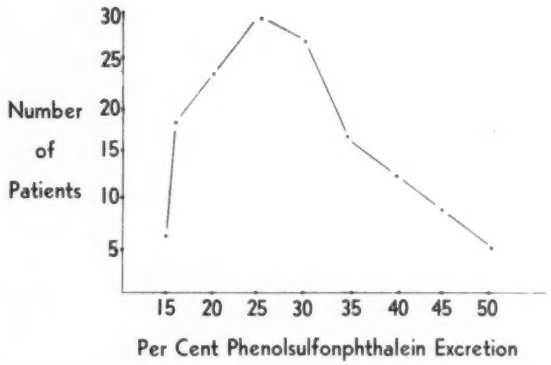

FIG. 1. Fifteen minute phenolsulfonphthalein determinations in 194 patients showing density of the contrast medium within the renal pelves on the five minute intravenous pyelogram greater than the roentgenographic density of the ribs.

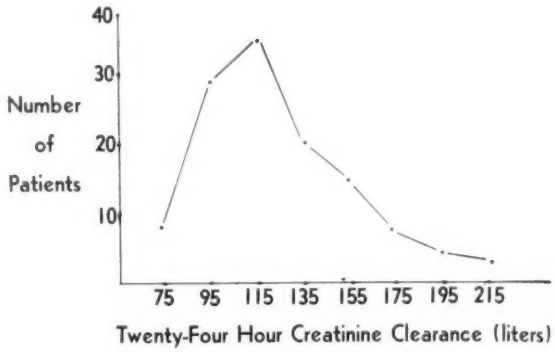

FIG. 2. Twenty-four hour creatinine clearance determinations in 194 patients showing a density of the contrast medium within the renal pelves on the five minute intravenous pyelogram greater than the roentgenographic density of the ribs.

liters. None of the patients who displayed pyelographic density at five minutes exceeding that of the ribs were found to have poor renal function.

Although false positive results could be anticipated in the presence of marked osteoperosis, no such cases were encountered in the series. The possibility of false negative results is readily apparent in cases of ureteral obstruction, overhydration and poor bowel preparation; 5 such cases were reviewed.

Errors of roentgenographic technique such as overexposure and underexposure proved to be of no consequence since both the density of the pyelogram and of the ribs were equally affected.

CONCLUSION

A method is offered for the utilization of intravenous urography for determination of renal function. Satisfactory renal function may be assumed if the density of the contrast medium within both renal pelves on the five minute intravenous pyelogram exceeds the roentgenographic density of the ribs. When utilized in this manner, excretory pyelography can simultaneously provide appraisal of both the anatomic and physiologic status of the kidney.

Unless the disease process necessitates the use of more sensitive measurement of renal function, on the basis of these objective findings it would appear unnecessary to perform other kidney function tests when the described pyelographic criteria are fulfilled.

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CLINICAL EXPERIENCES IN EXCRETORY PYELOGRAPHY*

A COMPARATIVE STUDY OF DIFFERENT CONTRAST MEDIA (HYPAQUE 50%, MIOKON 50% AND UROKON 50%) IN 3,454 CASES

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SINCE the development of urokon by Wallingford *et al.* in 1951,[28,29] several variations of the basic molecular structure have been introduced in attempts to provide a pyelographic medium which would produce better roentgenographic results with a decreased incidence of side reactions. The three compounds studied were used routinely in the pyelographic unit of the University Hospital. The results obtained with two of these compounds, hypaque 50 per cent (3,5-diacetamido-2,4,6-triiodobenzoic acid) and miokon 50 per cent (3,5-dipropionylamino-2,4,6-triiodobenzoic acid), in a series of 3,030 cases have been compared with a previous series using urokon 50 per cent (3-acetylamino-2,4,6-triiodobenzoic acid).

METHOD

The series consisted of the unselected and essentially uninterrupted patient complement (outpatients and inpatients) undergoing excretory pyelographic examination for any reason. The drugs were alternated weekly. Preparation of the patients included fluid restriction for a period of twelve hours prior to examination, but no purgatives or enemas were used. The minimum sequence of roentgenographing included a pre-injection exposure and a five, ten and fifteen minute postinjection series. Pyelographic quality was determined by evaluating the complete set of routine roentgenograms.

Injection times of forty-five to sixty seconds for an adult dose of 25 ml. to 30 ml. were consistently used for all media; appropriate adjustments in the volume of the drug and the injection rate were applied to children. The injection time was in effect the time required to comfortably inject the stipulated volume of the medium under sustained moderate pressure through a No. 20 or No. 21 gauge hypodermic needle. While some investigators[6,9,13,19,27] have maintained that rapid injection rates tend to increase the incidence of reactions, others[1,12,25,26] have not considered this an important factor in the production of reactions. Root and Strittmatter[21] indicated that slower rates might tend to promote reactions.

Allergic testing (intradermal and intraocular) was not carried out since past experience with these procedures has failed to provide reliable criteria for withholding or administering these drugs. Other investigators[2,9,10,11,14,24] record similar experiences. Intravenous testing has not been demonstrated to provide any more adequate control.[5,11,14,18] Histories of allergy have been reported[9,12,14,19,21,24,27] to correlate more satisfactorily with reaction incidence than testing procedures, but positive allergic histories provided no index as to the severity or type of reaction which resulted and was of little value in predicting a reaction in the individual case. One group of investigators[20] reported no statistical difference between allergic and nonallergic groups as to incidence or type of reaction. No screening on the basis of allergic history was employed in this series, and no antihistaminic drugs were administered prophylactically, although there is evidence indicating that this is probably a worthwhile procedure.[2,7,15,24]

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In studies with hypaque[1–4,6,9,10,13,20–23,26,27] and miokon,[2,3,18,22] wide variations in the incidence of reaction are reported. With reference to hypaque, for example, the incidence of reactions ranged from 5.5 per cent[10] to 100 per cent.[20] Similar comparisons of urokon data[4,16,23,25] are in closer agreement.

In this study "reaction" is defined as any change, apparent to the observer or obtained through questioning the patient, which developed after the injection of the drug. Reactions were observed, solicited and recorded by members of the Urological service who were assigned to the pyelographic unit. Periodic computations of reaction incidence throughout the term of the study revealed consistently recurring rates for total reaction incidence and specific reaction incidence when chronologically distinct portions of the series were compared. No attempt has been made to differentiate between allergic and nonallergic (psychogenic, neurovascular, autonomic, vasomotor) reactions. Although such analyses have been reported,[9,10,14,24] agreement on criteria for classifying reaction types has not been forthcoming.

Comparison of the roentgenographic results from various studies reveals a high degree of correlation. Most authors report very satisfactory results with hypaque[1,3,4,5,9,13,21,26,27] and miokon[2,3,18] and good results with urokon,[4,22,23,25] although various methods for assessing quality were used. In the current series the following standard was adopted: (1) excellent—pyelograms equal in quality and diagnostic value to those obtained by the retrograde method; (2) good—pyelograms not equal in quality to those obtained by the retrograde method, but of sufficient diagnostic value to render a retrograde study unnecessary; (3) fair—pyelograms showing partial delineation of structure but not sufficient for unequivocal interpretation; (4) poor—pyelograms showing some excretion of contrast material only; and (5) nonvisualization. Classification of pyelograms was made by a member of the senior Urologic staff at the time of the clinical interpretation of the roentgenograms for diagnosis. In the urokon series no attempt was made to differentiate between the "excellent" and "good" classifications of the hypaque and miokon series. Table III, therefore, indicates the total percentage of these two classifications in the hypaque and miokon series for comparison with the urokon data in which the "good" classification represents this total. In general, the method of preparing patients, administering the contrast material, interpreting reactions and assessing pyelographic quality is the same as that used in studies previously reported from this clinic.[16,17]

RESULTS

A total of 3,454 patients underwent examination in the course of this study; 1,665 patients received hypaque, 1,365 received miokon and 424 received urokon. The incidence of patients manifesting one or more reactions following the injection of hypaque was 29.4 per cent, following miokon 48.5 per cent and following urokon 58.0 per cent. The over-all reaction incidence is listed in Table I. In analyzing the breakdown of reactions with each medium (Table II) it is noted that, while the incidence of nausea with hypaque (11.0 per cent) and miokon (14.6 per cent) is essentially the same, the two- to threefold incidence of nausea (39.0 per cent) in the urokon series is significant. The principal differences in

TABLE I

INCIDENCE OF PATIENT REACTIONS TO PYELOGRAPHIC MEDIA

Medium	No. of Patients Receiving the Drug	No. of Patients Manifesting One or More Reactions	Per Cent of Patients Manifesting One or More Reactions
Hypaque	1,665	489	29.4
Miokon	1,365	663	48.5
Urokon	424	249	58.0

the incident reactions of hypaque and miokon are in relation to taste and flushing. A similar reaction pattern has been reported in another comparative study of these two media.[3]

One severe anaphylactoid reaction occurred in a patient who received miokon. This patient had undergone excretory pyelographic follow-up examinations on many occasions over a period of years. Over this period of time, a variety of media was used but none of those employed in the present study had ever been used before. There was no history of a previous reaction to contrast material. Another patient who had reacted severely to urokon 70 per cent had no manifest reaction to hypaque. This failure of cross sensitivity response with contrast media has been noted by others.[11,14]

Both hypaque and miokon yielded a high percentage of satisfactory roentgenographic results, together with a consistency of distribution within the various classifications, which rendered them virtually equal in roentgenographic value. Urokon did not produce as satisfactory results as either of these two media (Table III). Since no adjustment was made in the series for patients with impaired renal function, the group of cases in which the renal tract was not visualized represents a type of negative control. The near identical incidence of nonvisualization with the three media would tend to confirm the probability of random distribution within the series. Thus the possibility that the results obtained with a particular medium might be prejudiced by a disproportionate number of cases with impaired renal function is minimized.

Table II

DISTRIBUTION OF TYPES OF REACTIONS TO PYELOGRAPHIC MEDIA

Type of Reaction Occurring in Patients	Per Cent Occurrence Rate of Reaction Types		
	Hypaque 50 per cent	Miokon 50 per cent	Urokon 50 per cent
Nausea	11.0	14.6	39.0
Vomiting	2.9	4.3	6.2
Taste	8.0	21.9	6.5
Flushing	6.4	11.4	14.8
Warmth	2.2	2.2	14.9
Dizziness	2.0	4.3	4.9
Urticaria	1.6	1.6	3.7
Vein spasm	.8	.2	3.1
Sneezing	.2	.6	1.2
Choking	—	.6	—
Tingling	.6	.1	.9
Dyspnea	.1	.6	—
Cramping	.1	—	2.2
Chilling	—	—	1.9

Table III

ANALYSIS OF ROENTGENOGRAPHIC QUALITY

Degree of Roentgenographic Quality	Per Cent Distribution of Examinations		
	Hypaque 50 per cent	Miokon 50 per cent	Urokon 50 per cent
Excellent	5.9	7.5	58.5
Good	78.9 (84.8)	74.3 (81.8)	
Fair	11.7	14.1	31.1
Poor	1.8	2.3	9.2
Nonvisualized	1.7	1.8	1.3

SUMMARY

During the routine clinical use of 50 per cent hypaque sodium, 50 per cent miokon sodium and 50 per cent urokon sodium in 3,454 patients, the pyelographic quality was found to be essentially equal when hypaque and miokon were compared. Reaction incidence of these two media was also similar except for an increased incidence of unpleasant taste and flushing encountered with miokon. The clinical results obtained with both of these media are considered superior to those obtained with 50 per cent urokon sodium.

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CALCIFIED ADRENAL CYSTS*

By A. J. PALUBINSKAS, M.D., W. R. CHRISTENSEN, M.D., J. H. HARRISON, M.D., *and* M. C. SOSMAN, M.D.†

ADRENAL cysts are a rare and unusual pathologic entity. At the present time total case reports of this disease number approximately 80. Despite this fact there are only 3 published instances where a correct preoperative diagnosis has been made on the basis of roentgen findings. In 2 of these, the most important feature was thin laminar calcification of the cyst wall. The following is a report of 4 additional cases of benign adrenal cyst. All show this characteristic calcification.

It is hoped that this report will focus additional attention on this unusual entity as well as emphasize its consideration in the differential diagnosis of obscure abdominal masses.

REPORT OF CASES

Case 1. B.M. This fifty-five year old male boilermaker was referred to the North Bench Veterans Administration Hospital, Salt Lake City, Utah, for investigation of a calcified mass in the left upper quadrant. The patient's only complaint was moderate pain in the right hip and low back.

Past history was not remarkable. The patient denied trauma to the abdomen or back. No history suggestive of renal or endocrine malfunction could be elicited. There had been no intimate contact with sheep or dogs.

Physical examination revealed an apparently healthy male with normal vital signs and blood pressure. There were no significant positive findings.

Routine laboratory studies were not remarkable.

A chest roentgenogram was normal. Plain roentgenograms of the abdomen revealed a left upper quadrant soft tissue mass which impinged on and distorted the left renal shadow. It measured 10.5 cm. in greatest diameter and was outlined by a thin lamina of calcific density. Intravenous urograms showed good function bilaterally with normal calyceal architecture on the right. The left superior calyx showed a typical, extrinsic pressure deformity (Fig. 1) in association with the previously noted soft tissue mass. Retroperitoneal air studies clearly indicated that the mass was separate from, but displaced, the left kidney inferiorly and anteriorly.

A diagnosis of benign adrenal cyst was made by the radiologist.

At operation the left upper quadrant mass was found to be a grayish-white, translucent cyst containing gray-yellow, opaque fluid. It indented the superior pole of the left kidney,

Fig. 1. Case 1. The left adrenal mass is outlined by interrupted peripheral thin curvilinear calcification.

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† Deceased.

but was not part of it. The cyst was attached to the anterolateral margin of the left adrenal gland in the region of an orange-colored nodule of adrenal tissue. Cortical tissue was irregularly distributed over the surface of the cyst. The cyst measured 10×10.5 cm. and the nodule 3×3.5 cm. Both the cyst and the nodule were removed.

Gross examination showed a collapsed cystic structure weighing 15 gm. Its external surface was studded with nodules of cortical tissue. Calcific plaques could be palpated in the wall. When filled with saline, the cyst weighed 220 gm. (Fig. 2).

Microscopic study revealed the cyst wall to be composed of thick connective tissue. There were collections of cortical tissue on the external surface and calcific plaques on the internal surface. The cortical cells were distorted by stretching and compression. The internal surface of the cyst showed irregular collections of macrophages but no lining membrane. The nodule was composed of well-differentiated cortical tissue. There was no evidence of malignancy.

Final pathologic diagnosis was benign adrenal cyst with calcification and adrenal cortical adenoma.

Eight months postoperatively the patient was well except for continued hip and back pain attributed to degenerative osteoarthritis.

CASE II. H.B. This fifty-four year old housewife was admitted to the Heywood Memorial Hospital by Dr. O. J. Marcil with the provisional diagnosis of chronic gallbladder disease. Her chief complaint was dull pain present in the right upper quadrant and in the back just below the angle of the right scapula. The pain was intermittent, being brought on by exercise and heavy foods.

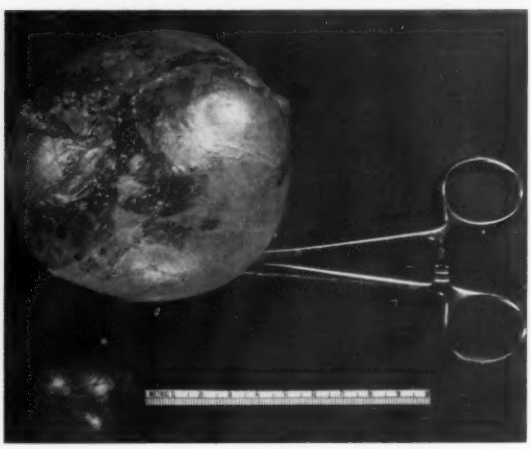

FIG. 2. Case I. Gross appearance of the adrenal cyst and adrenal cortical adenoma.

Past history was noncontributory. There was nothing to suggest urinary or endocrine abnormalities. The patient denied significant trauma to abdomen or back.

Physical examination revealed an apparently healthy female in no acute distress. Vital signs and blood pressure were normal. The only positive findings were tenderness in the right upper quadrant and a questionably palpable mass in the same region.

Laboratory studies showed no unusual findings. Hemogram and routine blood chemistries were within normal limits. The urine showed one to two white blood cells per high power field.

Roentgenographic studies of the chest, gallbladder, stomach and large bowel were likewise within normal limits. Plain roentgenograms of the abdomen showed a soft tissue mass measuring 10 cm. in diameter superimposed on the liver shadow (Fig. 3). Its inferior border was demarcated by a thin lamina of calcific density. The inferior half of the right kidney was identifiable below the mass, but there was no apparent displacement of this structure. Retrograde urography after retroperitoneal injection of air demonstrated that the mass produced a pressure defect on the superior pole of the right kidney (Fig. 4).

The possibility of a hydatid cyst of the liver was raised on the basis of the roentgenographic studies. The preoperative diagnosis was tumor of the superior pole of the right kidney.

At operation the right adrenal was found to be replaced by a blue-tinged cystic mass, 10 cm. in diameter. This was removed.

The gross specimen consisted of an irregularly shaped, cystic mass measuring 9×6×5.5 cm. Its surface was membranous and showed irregular areas of gray-white thickening, yellowish plaques up to 1.5 cm. in size and other small masses suggesting fat. On section the wall was found to consist of firm, gray-white tissue averaging 2 mm. in thickness. Areas of fine granularity and widely dispersed calcific plaques measuring up to 0.5 cm. in size were irregularly distributed throughout the wall.

Microscopic study showed the cyst wall to be composed of dense fibrocollagenous tissue showing areas of hyalinization and calcification.

Embedded in the wall were nests of typical adrenal tissue. A sparse lymphocytic infiltration was present. There was no internal lining membrane seen.

Routine check eleven months after surgery found the patient in good health without evidence of urinary or endocrine disturbance.

Case III. H.S. This forty-six year old white male salesman was admitted to the Peter Bent Brigham Hospital (#1E688) by Dr. James P. O'Hare for investigation of a tumor in the left adrenal area. He was essentially asymptomatic.

Past history was remarkable in that the patient had been refused life insurance three years before admission because of hypertension. Thereafter periodic examination had shown a blood pressure ranging from 140/90 to 160/104. Immediately before admission, a mass had been palpated in the left flank. An intravenous urogram had shown good renal function bilaterally, but demonstrated a 7 cm. soft tissue mass in the left upper quadrant. The kidney was displaced downward. The periphery of the tumor showed thin, curvilinear calcification.

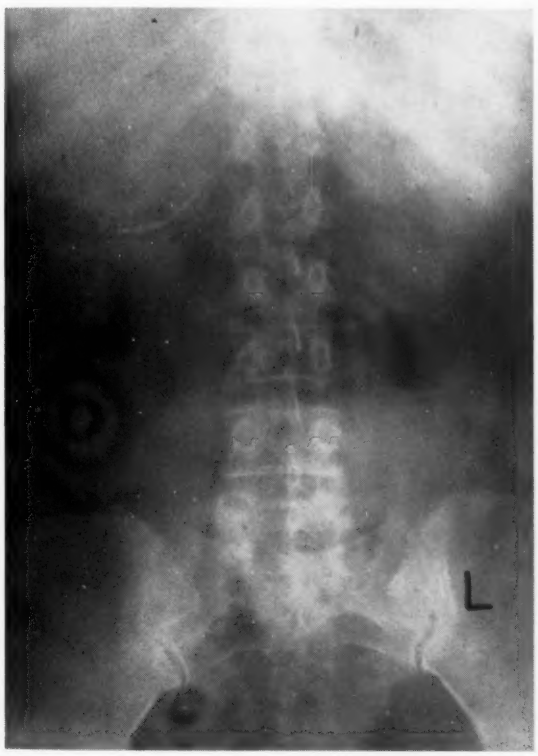

Fig. 3. Case II. The right adrenal mass is outlined by thin peripheral curvilinear calcification. Although an oral cholecystogram showed no definite evidence of gallstones, their presence is suggested by the cluster of faint densities just below the right adrenal mass.

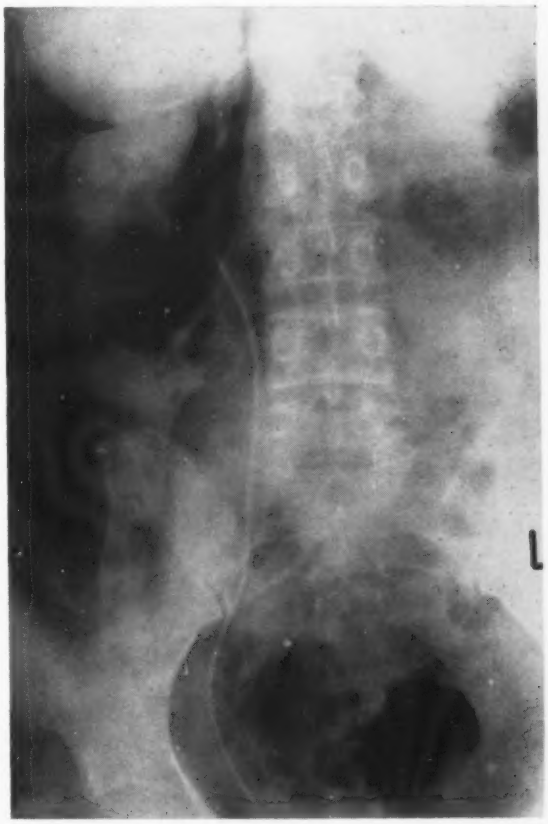

Fig. 4. Case II. Retroperitoneal air injection with catheter in place in the right ureter, its tip in the region of right renal pelvis. The pressure deformity of the superior pole of the kidney by the calcified adrenal mass is shown.

Physical examination revealed a healthy adult male in no distress. The only significant positive findings were a palpable, nontender mass in the left flank and separate blood pressure readings of 152/100 and 146/98.

Routine blood and urine studies were not remarkable. Renal function and benzodioxane tests were negative. The twenty-four hour 17 ketosteroid excretion was within normal limits as was an ACTH eosinophil response test.

Retrograde urography confirmed the presence of a peripherally calcified, soft-tissue mass which displaced a normal appearing left kidney inferiorly (Fig. 5). The preoperative diagnosis was tumor of the left adrenal gland.

Exploration revealed a baseball-sized cyst involving the left adrenal gland. The cyst was surmounted by a mass of atrophic adrenal tissue about one-third the size of a normal

FIG. 5. Case III. Preliminary roentgenogram taken prior to injection for retrograde urography. The displacement of the left kidney is shown. Arrows are opposite faintly outlined peripheral curvilinear calcification in the wall of the adrenal cyst.

gland. Flattened irregular clumps of adrenal tissue were scattered over the surface of the cyst. The cyst and adrenal gland were removed in one piece.

Gross inspection revealed a cyst measuring 5 cm. in greatest diameter plus a small remnant of adrenal gland containing a 1 cm. nodule. The surface of the cyst was yellow-gray in color and roughened with tags of adipose and fibrous tissue and flat yellow plaques. The cyst wall was fibrous in character and varied from 1 to 5 mm. in thickness. Hard, yellow, calcific material was irregularly distributed throughout the wall, in some areas involving its entire thickness.

Microscopic examination (Fig. 6) showed the cyst wall to be composed of dense hyalinized fibrous tissue which contained adrenal tissue undergoing varying degrees of compression. The adrenal cells contained large cytoplasmic vacuoles. No definite internal lining membrane was present. Areas of calcification, some of which were nodular, were irregularly distributed throughout the wall. The yellow plaques noted grossly on the external surface contained adipose, fibrous and adrenal tissue. No smooth muscle fibers were seen.

Final pathologic diagnosis was benign cyst of adrenal gland, probably of vascular origin, and adrenal cortical adenoma.

CASE IV. E.K.F. This fifty year old white married housewife, mother of 3 children, was admitted to the Peter Bent Brigham Hospital (#9K842) by Dr. J. P. O'Hare for evaluation of vascular hypertension and "shaking spells" of one month's duration. In addition to the above, the patient was known to have chronic pyelonephritis, carious teeth and psychiatric difficulty characterized by afternoon depression, fatigue and worry. During the year before admission, she had been having daily shaking spells. These began in the morning and were followed by a falling sensation. After the attacks, she was so exhausted that she had to rest the remainder of the day. Three weeks before admission she had developed pyuria accompanied by minimal urologic symptoms and was put on chloromycetin.

Past history was not remarkable.

Physical examination showed a well developed and nourished female, "poker-faced," but in no acute distress. Heart, lungs and abdomen were negative. There were numerous carious teeth. Blood pressure was 176/100.

Laboratory studies showed the blood to be normal. The urine was loaded with white blood

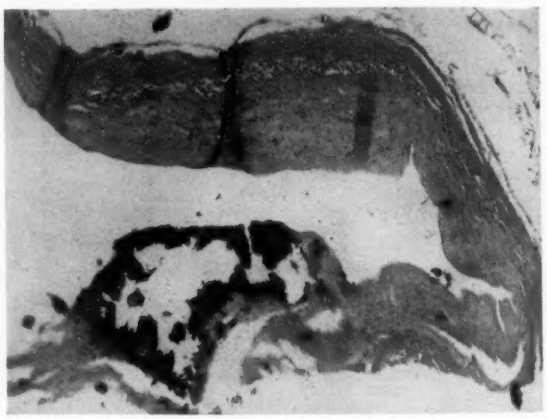

FIG. 6. Case III. Histologic section through the adrenal cyst wall showing dense hyalinized fibrous tissue, adrenal cortical cell deposits, and areas of calcification (×35).

cells. *E. coli* and *Staphylococcus aureus* were cultured from the urine. Phenolsulfonphthalein excretion was 15 per cent at fifteen minutes, 20 per cent at thirty minutes and 70 per cent at two hours. The twenty-four hour excretions of 17 ketosteroids and 17 hydroxycorticoids were within normal limits.

Roentgenograms of the abdomen demonstrated an ovoid, soft tissue mass 6 cm. in greatest diameter in the region of the superior pole of the left kidney. The mass showed laminar, curvilinear calcification at its inferior border and also on the circumference at 1 and 2 o'clock. Intravenous pyelography revealed good concentration bilaterally. There were mild changes consistent with pyelonephritis bilaterally. In addition, the intrarenal structures on the left showed minimal deformity and rotation due to a soft tissue mass in the region of the superior pole (Fig. 7).

Laminagraphy was performed. The mass was well demonstrated at the 8.5 cm. level. It measured 6.5×5 cm. and was definitely distinct from the kidney. Spotty curvilinear calcifications were demonstrated within the mass.

The final preoperative impression was benign calcified cyst of left adrenal.

At operation a cystic, soft tissue mass measuring 6 cm. in diameter was found lying above the left kidney and against the posterior wall of the thorax. This burst during the operation and a cloudy fluid was expelled. The cyst and some contiguous adrenal tissue were excised.

Gross pathologic examination revealed an ovoid cyst weighing 10 gm. and measuring 6×4.5×4.5 cm. Its shiny, glistening wall varied in thickness from 0.1 to 1.5 cm. and was studded with irregular yellow plaques. Some of these were calcified. Attached to the surface of the cyst was a fragment of bright yellow adrenal tissue measuring 7×3×0.5 cm. When sectioned, this was seen to be composed of layers of cortical tissue with a thin layer of medullary tissue interposed, thus indicating that the adrenal gland had been displaced and compressed by the cyst (Fig. 8).

The wall of the cyst was composed of dense, hyaline connective tissue with no inner epithelial lining. Numerous calcified areas were present. The adrenal tissue was confined to one portion of the wall, appeared thin and compressed and was composed exclusively of cortical cells. Final diagnosis was benign adrenal cyst with fibrous and focally calcified wall.

Fig. 7. Case IV. Intravenous urogram demonstrating slight deformity of the superior calyx of the left kidney. The suprarenal mass is sharply outlined, in part by the surrounding fat and in part by the curvilinear calcification in its wall. The mass appears oval as if compressed between the spleen above and lateral, and the kidney inferiorly.

DISCUSSION

The 4 cases presented above appear to be examples of a clear cut pathologic and roentgenologic entity, namely benign cyst of the adrenal gland. The first such case was reported by Greiselius[19] in 1670 and a second was added by Rayer[37] in 1837. Recent reviews of the world literature have indicated a total of 50 cases up to 1947 and 81 cases up to 1955. This unusual rarity has been questioned by Wahl[49] who found 9 unsuspected cysts in approximately 4,000 autopsies. One might surmise that the paucity of reports in the literature is a manifestation of their clinical silence rather than true rarity.

FIG. 8. Case IV. Photograph of the collapsed empty cyst with laminated walls and attached tissues.

In general, as with our cases, presenting complaints have been noncharacteristic and would not lead to consideration of such a diagnostic possibility. The list includes nausea and vomiting, abdominal, back or flank pain, palpable adbominal mass, abdominal enlargement, indigestion, flatulence, etc. Only a few of the reported cases have had a suggestive history of antecedent trauma.[14,16,18,33] It seems likely that such specific symptoms as do occur result from hemorrhage into the cyst or can be attributed to the actual bulk of the lesion.

In most instances, clinical interest has been aroused by the palpation or roentgenologic demonstration of an abdominal mass.

The difficulties of differential diagnosis are obvious and have been discussed at length.[3,15,17,18,26] Cysts and tumors of spleen, pancreas, liver, kidneys and mesentery as well as empyema of gallbladder, splenic artery aneurysms, dermoid cysts, hydatid cysts, chylous cysts, urachal cysts and solid adrenal tumors must be considered. Localization to the adrenal region is a major step in differentiation. Displacment of the kidney with accompanying distortion of the superior collecting structures is a significant localizing indication which occurs in a majority of the reported cases. Nevertheless, even exact restriction to the adrenal area still leaves the diagnosis in doubt.

The presence of calcification is a major roentgenologic sign. Microscopic calcification occurred in roughly one-third of the cases cited in the English literature and in one-sixth of these the density was visible roentgenographically. When calcification is peripheral and curvilinear, the diagnosis of benign adrenal cyst is clearly suggested. As in our cases, other authors have made the preoperative diagnosis on the basis of these findings; namely, that of a mass localized to the adrenal region, the periphery of which is outlined by curvilinear calcification.[25,42]

Obviously, either adenoma or carcinoma of the adrenal may be apparent in plain roentgenograms as soft tissue masses in the suprarenal area. The soft tissue density will not serve to distinguish between these lesions. Calcification, if present, may be helpful. The peripheral curvilinear calcification of the cyst is fairly characteristic. Calcification, which is rather rare in the solid tumor, generally appears as scattered nodular areas of opacity within the substance of the soft tissue mass, but may localize as a peripheral layer and give an appearance similar to that of the cyst. In such cases the differentiation can usually be made on the basis of clinical endocrinologic changes. Excretion of 17 ketosteroids and 17 hydroxycorticoids in the patient having a cyst is either normal or subnormal, in contrast to the elevation caused by adenoma or carcinoma. Under ACTH stimulation the excretion of these same compounds will be within normal limits in the presence of a cyst, whereas the adenoma

exhibits a further elevation of excreted steroids and the increased level caused by carcinoma generally shows no further rise in response to this physiologic stimulus.

The above findings have certain implications insofar as therapy is concerned. Treatment clearly consists of surgical removal. Since minimal endocrine abnormalities have been present, this approach must be justified on the basis of accuracy of diagnosis and relief of local symptoms. If the other adrenal is assumed to be intact, one would not hesitate to sacrifice the adrenal remnants on the affected side. There is some question that this is invariably correct. Bilateral cysts have been reported.[13,25,28,30,31,47,49] These occur in approximately 15 per cent of cases. If the function of both adrenals has been compromised, removal of the cyst and residual gland may throw a patient into an Addisonian crisis. This appears to have been a possible cause for the death in postoperative shock of the brother of one of our patients. Such considerations should lead to careful investigation of the possibility of bilateral cysts as well as their occurrence in other members of families in which they have been demonstrated.

The general clinical and pathologic characteristics of these cysts are well known. The incidence is 50 per cent higher in females than in males. They occur in about equal numbers on right and left sides. The majority are discovered in the thirty to sixty year age group, but they have been reported in all age groups from two months to seventy years.[1,9,37,40,47,50] Reported size has varied from that "of a boy's marble" up to 30 cm. in diameter. The cysts may be uni- or multilocular and the contents vary from translucent fluid to dark gelatinous material mixed with tissue debris. The wall components are generally dense fibrous connective tissue which may be hyalinized or rarely show amyloid degeneration. The thickness of the wall is generally 1–5 mm. though some measuring 1 cm.[15] and 3 cm.[18] have been reported. A variety of other cellular elements have been reported as components of the wall.[7,28] As has been noted previously, one-third of the cases noted in the English literature show calcification microscopically and in one-sixth this calcification is visible roentgenographically.

A considerable variety of pathologic classifications of adrenal cysts have been suggested.[21,23,25,45] As would be expected from the number proposed, none is completely acceptable. Some authors consider classification of little importance since the exact nature of the cyst is not determinable in most instances. For the sake of completeness, we have included that of Terrier and Lecene,[45] the one most generally accepted. This is as follows: (1) True glandular cysts; (2) parasitic cysts; (3) cystic adenomas; (4) serous (lymphatic) cysts; (5) pseudocysts.

The possible etiology, microscopic appearance, and relative frequency of the above cysts have been discussed in numerous articles.[15,23,25,29,30,33,38,41,48] Little agreement is apparent, but some general conclusions may be stressed.

Serous (lymphatic) cysts arising from lymphatic structures and pseudocysts secondary to hemorrhage and/or necrosis or cystic resolutions of hematomata in the absence of adrenal tumors are the most common types of cysts.

The true incidence of cystic adenomas is obviously unknown. Published opinion varies from "may occur"[15,38] to "occur rarely"[18,29,48] to "significant number of definite cases."[6,10,18,22,25,49,52]

Reports alleging true glandular cysts of the adrenal are extremely rare.[20,25,39] Some authors feel that with the exception of congenital occurrence, true retention cysts probably do not occur in man.[15,31,38,43,44,48,52]

Parasitic cysts of the adrenal are uncommon. Barnett[5] noted a 0.5 per cent incidence of echinococcus cysts of the adrenal in patients with this same disease elsewhere.

The over-all situation appears even more confusing when one realizes that the above classification includes neither cysts of vascular origin nor those occurring in the adrenals as part of the generalized involve-

ment in polycystic disease of kidney and liver.[12,24,25,46]

SUMMARY

Four cases of benign adrenal cyst have been presented. None of the patients had demonstrable hormonal abnormalities. In each instance roentgenographic examination showed peripheral curvilinear calcification in the cyst wall.

Although not absolutely characteristic, the presence of thin laminar calcification at the periphery of a soft tissue mass in the suprarenal area is strongly suggestive of a benign adrenal cyst.

The two important conclusions which can be made from proper roentgenologic examination, prior to operation, if the findings are as noted above, are: (1) that the demonstrated tumor is cystic and benign; and (2) that it arises in or from the adrenal gland.

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FURTHER EXPERIENCES WITH THE RADIOISOTOPE RENOGRAM*

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THE radioisotope renogram, a clinical test of individual renal function, was performed first in May, 1955.[4] The test consists of injecting a tracer dose of I^{131}-labelled contrast medium intravenously and then recording continuously the level of radioactivity emanating from each kidney region by means of gamma-ray scintillation probes connected to recorders through rate meters (Fig. 1). Urokon (sodium acetrizoate) I^{131} was used initially as the test agent. It did not produce characteristic tracings and was found lacking in usefulness as a test material. Diodrast (iodopyracet) I^{131} was tried next and was found to produce a distinctive pattern for the normal as well as the abnormal kidney. The three segments of the normal pattern represent renal vascular capacity, tubular cell function and drainage of urine from the kidney (Fig. 2). Following this discovery, four other I^{131} labelled contrast media were tested. NaI^{131} is filtered passively by the renal glomeruli and gives a graph similar to urokon. Hypaque (sodium diatrizoate), miokon (sodium diprotrizoate), and renografin† (methylglucamine diacetylaminotriiodobenzoates)[6] are secreted by the renal tubules and produce tracings similar to diodrast, although they pass through the kidney less rapidly. However, they possess one practical advantage. They are not removed from the blood by the liver, which extracts 10–15 per cent of diodrast with the production of an occasional false right renogram.

FIG. 1. Test equipment (see text).

TECHNIQUE

The method of performing the test has not changed materially since originally described.[4] It is apparent, however, from reports of investigators elsewhere[1,2] that considerable difficulty is being encountered in obtaining satisfactory results. Therefore, each step of the technique that is important for success is reviewed.

1. An upright roentgenogram of the abdomen must be made prior to the test for precise location of the kidneys. While it would seem obvious that the counters must be "looking" at the kidneys in order to obtain an accurate renogram, some have left out this step and guessed the location of the kidneys with misleading results. Occasionally, in infants or seriously ill patients, the prone position may be substituted.

2. The patient should be weighed just

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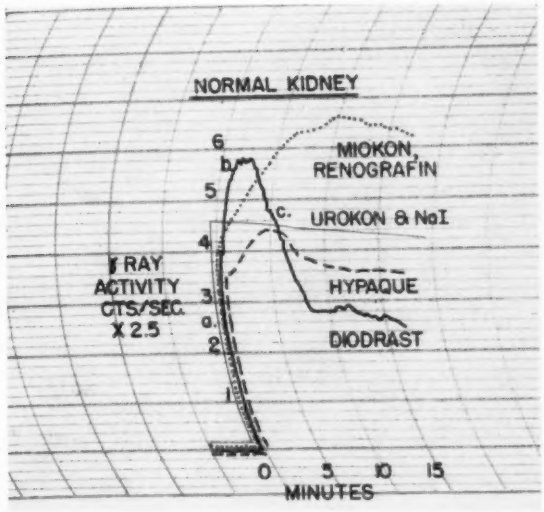

Fig. 2. Normal renograms, showing vascular spikes (a), tubular secretion phases (b), and drainage legs (c).

prior to the test in order to determine accurately the dose of isotope to be used. Each technician performing the test will find it necessary to compute a dosage schedule by performing a series of tests among normal individuals of various weights. Our present dosage range is 5 to 20 μc (average 1μc/6 kg.).

3. The scintillation crystals (1 inch cubes) in the probes should be heavily shielded and properly collimated. We use a minimum thickness of one inch of lead around the crystals except at the aperture (1 inch diameter) "facing" the kidneys. The crystals are withdrawn one inch within the lead shields. This reduces the amount of radiation detected from nonrenal tissue.

4. With the patient seated comfortably, the scintillation probes should be directed toward the posterior aspect of the kidneys, keeping the geometric factors equal on each side. With the use of hypaque (our preference at this time), miokon or renografin, it is not necessary to angle the probes downward (as in Figure 1), as emphasized previously with the use of diodrast, in order to avoid the liver. Fortunately, the radiation signal from the liver, aorta and vena cava can be reduced sharply by proper shielding and positioning of the probes, since the strength of the radiation signal received is inversely proportional to the square of the distance from the source.

5. The two rate meters must be calibrated properly so that each unit will produce an equal response. If attempted simultaneously by placing a single radiation standard at a chosen distance from the two probes, the results are usually inaccurate. We have found it necessary to place a small radiation standard at a measured distance from the crystal at the aperture of each probe. This is performed individually. It is not necessary to use I^{131}, since cobalt, antimony or radium work equally well. The radium dial of a wristwatch is a practical and constant source for this purpose. However, one must be certain that the rate meters are adjusted to give an equal response to the energy spectrum of the radiation source used when compared to I^{131}.

6. The speed with which the recorder responds to the signal should be moderately rapid during the initial vascular spike. Thereafter, the response may be slowed down to permit a more regular tracing.

7. During introduction of this test to technicians for the first time, a large number of tests should be performed among subjects with normal kidneys in order to become acquainted with the technique of testing, instrumentation problems, and to note various types of normal patterns.

In reviewing difficulties encountered by others with the test, one or more of the above steps were violated and could account for the discrepancies in their results.

Interpretation of the Renogram

Characteristically, the normal tracing (Fig. 2) has three segments which can be distinguished readily. The initial spike (a) represents the vascular capacity of the kidney plus that of background tissue. When a probe is placed over the area where a kidney has been removed, the spike is approximately one-third of that obtained when a normal kidney is present. This fact has been substantiated further by making renograms

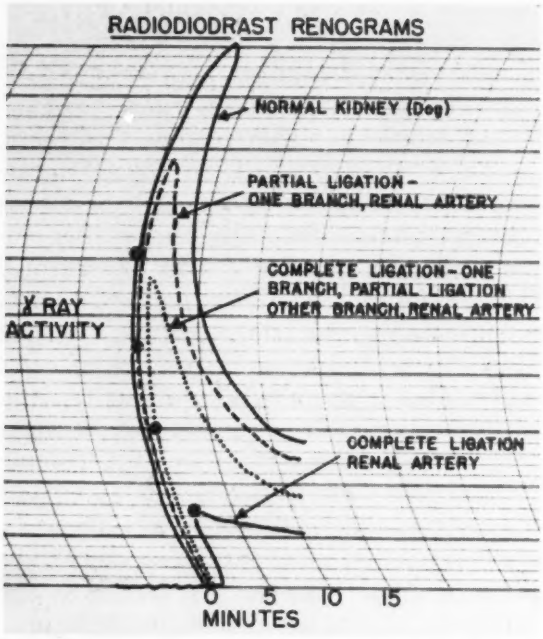
RADIODIODRAST RENOGRAMS
NORMAL KIDNEY (Dog)
PARTIAL LIGATION - ONE BRANCH, RENAL ARTERY
COMPLETE LIGATION - ONE BRANCH, PARTIAL LIGATION OTHER BRANCH, RENAL ARTERY
X RAY ACTIVITY
COMPLETE LIGATION RENAL ARTERY
0 5 10 15
MINUTES

FIG. 3. The diminution in the vascular spike and functional segments is proportional to the degree of renal artery ligation.

before and after ligation of the renal artery in dogs (Fig. 3).[7]

The secondary rise in the tracing (Fig. 2, b), beginning during the second or third minute and lasting three to five minutes, is due to tubular cell secretion of the contrast medium. Therefore, the steepness and height of the ascent is proportional to kidney function. This segment is a good index of the function of the whole organ (Fig. 4), and, furthermore, tubular cell activity is usually the first function to be disturbed in renal disease.* When a highly vascular area such as the lung or heart is placed under the probe, a sharp fall in the tracing occurs during the second phase and is due to clearance of the I^{131} agent from the vascular system by the kidneys. Of course, when the kidney is absent or nonfunctioning or the renal artery is blocked, a secondary ascent does not occur.

The third and final leg in the normal renogram (Fig. 2, c) is the descent in the tracing starting in the fifth to eighth minute and there is a rapid fall for about five minutes in an exponential fashion. This fall parallels the blood curve and represents evacuation of the radioactive urine from the kidney as well as a sharp decrease in background activity. If there is stasis of urine in the renal pelvis due to ureteral obstruction or reflux, the tracing will show less desent or an actual continued rise depending upon the degree of obstruction and function (Fig. 5).

* This applies particularly to hypertension.

RESULTS

We have now performed over 1,000 renographic studies among individuals with normal and abnormal kidneys and in animals. Results characteristic of a normal pattern, nonfunction, various degrees of dysfunction, and obstructive uropathy have been obtained. New light has been shed upon the action of the kidney during neurogenic and surgical shock. When neurogenic shock is elicited by venapuncture trauma, urine formation continues normally, but due to spastic constriction of the ureter there is stasis of urine in the renal pelvis for fifteen

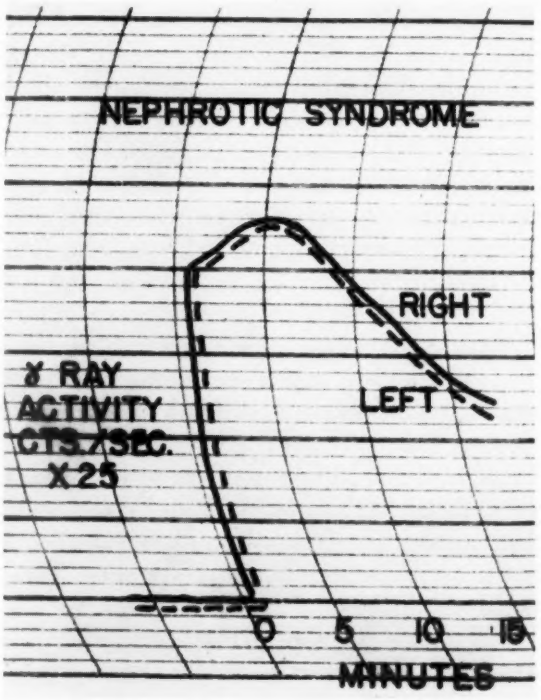

FIG. 4. When renal parenchymal disease is present, the vascular spikes are diminished and the functional segments are shorter and less steep.

to twenty minutes. Following this, the secondary leg (b), which had been rising steadily goes into a sharp decline (phase c) after release of the spasm. A renogram obtained during a major surgical procedure also revealed a continuation of the secondary rise, although no urine formation occurred. Apparently, the tubular cells retain their ability to secrete the test agent, although water is not excreted. These features, plus those differentiating anuria or oliguria caused by acute renal failure from obstructive uropathy will be presented in detail elsewhere. The clinical use of the test in our hands has been directed chiefly toward evaluation of the kidneys among hypertensive individuals.[5]

One hundred and four pateints with hypertension (diastolic blood pressure over 95 mm. Hg) have had renograms plus one or more standard individual renal function tests including excretory urography, blood and urine chemistry, renal clearance and dye tests, and aortography. Thirty-three were found to have bilateral normal renograms and standard renal tests and were spared extensive urologic investigation as to the cause of their hypertension. Forty-four were found to have bilateral abnormal renograms with a false positive rate of about 8 per cent. The remaining 27 had unilateral abnormal renograms with 2 false positive tests. The latter group was investigated extensively, urologically. Surgical correction of renal arterial defects or nephrectomy was performed in 15 patients, with good results in 7 during a follow-up period up to three years. These results will be discussed in more detail elsewhere. Thus, our results allow us to have confidence in the renogram, renal clearance and dye tests, and aortography as means of detecting unilateral renal disease. As yet, we do not know of a test which will indicate those patients with hypertension curable by nephrectomy or renal arterial surgery.

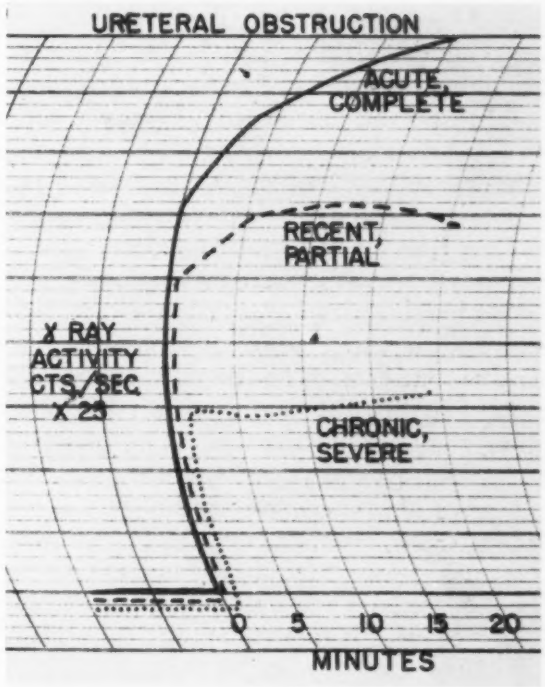

Fig. 5. In obstructive uropathy, the vascular spike and functional segment are decreased proportionately to the duration and degree of obstruction. The terminal leg rises or falls according to the degree of obstruction and function present.

DISCUSSION

To date, the results of the renogram in our hands have been qualitative only. Direct comparison of the right and left renogram furnishes the most valuable information. The production of a "ratio" tracing representing only the difference between the two renograms[1] is subject to misinterpretation, since artifacts in the individual tracings recognizable by experienced observers would remain undetected. The uncontrolled factor of distance between the kidney and body surface prevents quantitative determinations. Recent experiments in our laboratory have suggested means of overcoming this barrier to more precise measurement of renal functions externally.

The renogram does not displace standard methods of studying the kidney, such as individual renal clearance tests, urography, and arteriorgaphy. The latter two methods are indispensable as means of reaching a diagnosis, while the renogram can give only qualitative information concerning three functions of the kidney. The renogram does not reveal information that cannot be ob-

tained by utilizing all urologic methods of studying the kidney. But it will often reveal more than any one of the conventional renal tests, and more easily. We have confirmed abnormal renogram results in the face of normal excretory urograms and repeatedly have found the excretory urogram to be a poor index of renal function.

The renogram's main usefulness in our hands has been the evaluation of the kidneys and their arterial supply among hypertensive patients. The correlation of the renogram with the final diagnosis has been quite good. Thus, the finding of bilateral normal renograms would spare the patient extensive urologic investigation which involves cystoscopy, retrograde catheterization, and aortography. The avoidance of these hazardous and economically distressful procedures is further appreciated when one takes into account the high percentage of hypertensive patients having normal kidneys.

During the test the patient experiences only a small needle puncture and otherwise sits comfortably throughout the short procedure. Since only tracer amounts of both I^{131} (5–20 μc) and contrast medium (2–10 mg.) are used, no untoward reactions have occurred. The radiation hazard to patient and technician is not significant. In the individual with normal kidneys, 50–60 per cent of the I^{131} agent is excreted in the urine in thirty minutes.

SUMMARY

The radioactive isotope renogram is a useful qualitative test of individual renal function in a variety of medical and urologic kidney disorders. Its chief clinical value is as a screening test among hypertensive patients in order to find surgically correctable unilateral parenchymal or renal arterial disease.

Inaccurate results can be minimized by carefully following each step of the technique as has been outlined in detail.

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SOFT TISSUE ALTERATIONS IN ELBOW TRAUMA*

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EXTREMELY subtle, occult or subliminal fractures of the elbow joint nearly always leave an obvious, relatively specific soft tissue alteration. In 1954, Norell[7] indicated that certain extracapsular accumulations of fat described by the anatomists could easily be identified on routine roentgenographic examinations. An anatomic description of these specific soft tissues from "Gray's Anatomy" is as follows: "Between the capsule and the synovial membrane are three masses of fat: the largest, over the olecranon fossa is pressed into the fossa by the Triceps Brachii during flexion; the second over the coronoid fossa, and the third over the radial fossa, are pressed by the Brachialis into their respective fossae during extension." Of much more significance was the fact that on roentgenographic examination there was a consistent pattern for the normal elbow, and a fairly consistent alteration in the normal pattern in the presence of an intracapsular effusion. In pediatric patients, as the result of an intracapsular effusion caused by traumatic or inflammatory lesions, Norell demonstrated a specific displacement and visualization of the adipose tissue in the olecranon fossa at the elbow joint. With a history of trauma this alteration was most often due to fracture whether or not the fracture was demonstrated on the original roentgenogram.

NORMAL ROENTGEN FINDING

On roentgenographic examination the extracapsular fat accumulations are seen only, or to best advantage, in the lateral view of the elbow. The roentgen image finding corresponding to the adipose accumulations in the coronoid and radial fossae is a thin, roughly triangular radiolucency seen in close proximity to the ventral aspect of the distal humerus (Fig. 1). Of considerable importance is the fact that this shadow is a summation of the two fat pads and not a specific anatomic entity. Because of the shape of the condyles and the depth of the olecranon fossa, the dorsal extracapsular fat pad lies hidden ventral to the dorsalmost projections of the cortex of the condyles (Fig. 2). It is for this reason that the dorsal or olecranon fat pad is never seen in the lateral view of a normal elbow in spite of the fact that it is the largest of the extracapsular fat pads.

FIG. 1. Lateral roentgenogram of the left elbow demonstrating the characteristic appearance of the extracapsular fat pads. In this case there was no history of recent injury. The olecranon or dorsal fat pad cannot be seen on the dorsal aspect of the humerus.

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FIG. 2. Lateral roentgenogram of the left elbow of a fresh cadaver following injection of 5 cc. of contrast medium. The attachments of the capsule are demonstrated together with the depth of the olecranon fossa. Although not visible in the reproduction, the fat pad can be seen to be displaced on the original roentgenogram.

ALTERATIONS PRODUCED BY INTRACAPSULAR EFFUSION

As an intracapsular effusion develops within the elbow joint, the generalized increase in size results in an increase in the anteroposterior dimension of the posterior portion of the elbow joint space and a dorsal displacement of the olecranon fat pad. The dorsally displaced fat pad becomes visible on the lateral roentgenogram of the elbow when the displacement is such as to cause the fat pad to be projected free of the condylar cortex. Fortunately, only a very small effusion is necessary to produce this effect (Fig. 2 and 3). The dorsal extracapsular fat pad is visualized as a radiolucent shadow on the dorsal aspect of the distal humerus at about the same level as the ventral fat pad. The appearance of this radiolucency is generally crescentic, although considerable variability in length and thickness occur. The effusion also displaces the ventral extracapsular fat pad anteriorly in varying degrees, although this change is less constant and less predictable (Fig. 4). The shape of the capsule at the site of the extracapsular fat pad attachment is significant since the fat pads tend to assume the shape of the capsule as the effusion becomes larger. The anterior fat pad becomes inclined in an angular direction corresponding to the capsule. The dorsal fat pad tends to assume a curvilinear shape corresponding to the posterior bulge in the capsule (Fig. 3).

MATERIAL

Since Norell's[7] findings appeared useful in the routine interpretation of roentgenograms of the elbow, all the elbow roentgenograms taken in the departments of radiology at St. Michael's Hospital and Riverview Hospital between January 1, 1951, and December 31, 1957, were reviewed. All of the surveyed elbow roentgenograms were retained for this study with the following exceptions: (1) any roentgenogram taken through a plaster cast; (2) roentgenograms not taken specifically for the elbow joint, *viz.*, humerus and forearm; (3) roentgenograms taken with a portable apparatus in

FIG. 3. Same elbow as shown in Figure 2 following injection of an additional 5 cc. of contrast medium.

Fig. 4. (*A*, *B*, *C* and *D*) Lateral roentgenograms of the elbow demonstrating the variability in size, shape and position of the ventral and dorsal extracapsular fat pads produced by fractures.

the orthopedic room and not processed in the main department dark room; (4) any roentgenograms other than the original, with the exception of those from a series of roentgenograms which were felt to exhibit findings which might have a bearing on the atypical patterns of soft tissue alterations pertinent to this study. No case in this series had had a specific examination for soft tissue detail. All roentgenograms were reviewed and evaluated for the alterations in the soft tissue with special reference to

TABLE I

AGE DISTRIBUTION

Years	No.	Age (yr.)	No.	Age (yr.)	No.
0– 9	102	1	4	11	6
10–19	97	2	14	12	12
20–29	61	3	7	13	10
30–39	71	4	14	14	10
40–49	59	5	14	15	12
50–59	45	6	15	16	13
60–69	19	7	13	17	12
70–79	14	8	15	18	3
80–89	2	9	6	19	7
90–99	1	10	12		

Norell's findings. In addition to the usual visual inspection, all roentgenograms were studied with a 5× magnifying lens, and 5.4 reducing lens. All data were recorded on a mechanically sortable punch card (Key-Sort) by a hand operated punch, to facilitate accurate and rapid handling of data.

GENERAL INFORMATION

Four hundred seventy-one patients, comprising 193 females and 278 males, were roentgenographed, the total number of examinations amounting to 638. These included 99 examinations of the opposite normal elbow and 68 additional examinations which were made for evaluation of serial changes thought to have a bearing on abnormal or atypical original roentgenograms.

This latter group was not included in this statistical presentation since the arbitrary criterion for inclusion would produce considerable bias. The age distribution is shown in Table I by decades. A breakdown is also given for the age group from one year to nineteen years.

The number and types of fractures and dislocations are listed in Table II. Although a total of 170 fractures were demonstrated, 20 are not included in this series since they were associated with dislocation and were considered a part or complication of the dislocation.

SOFT TISSUE FINDINGS

Statistically, the soft tissue findings were considered in two groups: (1) those in the 99 opposite normal elbows and (2) those in the original roentgenograms of the 471 patients studied. In the second group the findings were broken down into three subgroups: (a) patients with fractures; (b) patients with dislocations; and (c) patients with neither fracture nor dislocation. The extracapsular fat in the radial and coronoid fossae was referred to as the ventral fat pad and simply labeled ventral in the tables; the extracapsular fat pad in the olecranon fossa was called the dorsal fat pad and was labeled dorsal. If a fat pad was visualized, it was said to be present and was indicated in the table by a plus sign; if it was not seen, it was said to be absent and was indicated by a zero.

All of the 99 opposite normal elbows examined had an identifiable ventral fat pad, and in no case could the dorsal fat pad be seen.

TABLE II

TYPES OF FRACTURES AND DISLOCATIONS

	No.
Fractures	
Supracondylar	55
Radial head	37
Olecranon	19
Coronoid process	12
Separation of lateral or medial epicondyle	11
Lateral condyle with separation of the capitellum	6
Bicondylar T or Y fractures	3
Lateral or medial condyle	3
Shaft of the humerus	3
Monteggia fracture	1
Total	150
Dislocations	
Anterior	2
Posterior	30
Medial	2
Lateral	1
Post reduction roentgenograms only	2
Total	37

Table III

SOFT TISSUE FINDINGS ON ORIGINAL ROENTGENOGRAMS

Ventral o	Dorsal o	39
Ventral o	Dorsal +	37
Ventral +	Dorsal +	102
Ventral +	Dorsal o	293

Soft tissue findings on the original roentgenograms are shown in Table III.

(*a*) *Soft Tissue Findings in Fractures.* Of the 150 cases in which a fracture was present, the soft tissue alterations were characteristic in 71, *i.e.*, the dorsal fat pad was displaced and visible and the ventral fat pad could also be visualized (Table IV). In 29 cases the presence of an intracapsular effusion was indicated by a posterior displacement and visualization of the dorsal fat pad, but the ventral fat pad could not be seen. Further, 20 cases, or 13 per cent, exhibited an atypical and abnormal pattern not demonstrated in Norell's series of cases. This consisted of the disappearance of the ventral fat pad and a failure to visualize the posteriorly displaced dorsal extracapsular fat pad in circumstances that apparently warranted its appearance. Thus, 120, or 80 per cent, of the fracture cases exhibited an abnormal soft tissue pattern of which 83 per cent were typical of intracapsular effusion. The remaining 30 cases, or 20 per cent, exhibited a normal soft tissue pattern. Five of these were extracapsular fractures which would not be expected to have an intracapsular effusion associated with them. Two occurred in elbows that were severely deformed by old healed supracondylar fractures and, because of the deformity, a fair sized effusion could easily be masked. In 3 other cases the technical quality of the roentgenograms was such that soft tissue evaluation was not satisfactory even though the fractures were demonstrated. This left only 20 cases, or 13 per cent, in which no obvious reason for the failure to develop an abnormal soft tissue pattern could be given, except, in general terms, that which will be indicated later in a discussion of the factors affecting visualization of the soft tissue and alterations in the presence of trauma.

Table IV

SOFT TISSUE FINDINGS IN 150 FRACTURE CASES

Ventral o	Dorsal o	20
Ventral o	Dorsal +	29
Ventral +	Dorsal +	71
Ventral +	Dorsal o	30

Table V

SOFT TISSUE FINDINGS IN 37 DISLOCATION CASES

Ventral o	Dorsal o	14
Ventral o	Dorsal +	5
Ventral +	Dorsal +	9
Ventral +	Dorsal o	9

(*b*) *Soft Tissue Findings in Dislocations.* The analysis of the soft tissue alteration in 37 cases of dislocation demonstrated greater incidence of atypical or abnormal patterns than did the fractures (Table V). Only 14, or 37 per cent, exhibited evidence of intracapsular effusion and 5 were atypical in that the ventral fat pad was not identified. In 9 cases, or 24 per cent, the soft tissue pattern was normal. In 14, or 37 per cent, neither ventral nor dorsal fat pads could be visualized.

(*c*) *Soft Tissue Findings in Remaining Cases.* In the group of cases exhibiting neither fractures nor dislocation only 30 of the 284 demonstrated an abnormal soft tissue pattern (Table VI). In addition to traumatic effusion the following conditions were noted: osteochondritis, osteochrondromatosis, osteoarthritis and rheumatoid arthritis. In view of the fact that most of the roentgenograms were taken before the

Table VI

SOFT TISSUE FINDINGS IN REMAINING 284 CASES

Ventral o	Dorsal o	5
Ventral o	Dorsal +	3
Ventral +	Dorsal +	22
Ventral +	Dorsal o	254

full implications of the soft tissue alteration were appreciated, it is reasonable to expect that an additional number of minor fractures might have been demonstrated by special views or techniques.

SIMILARITY OF FINDINGS

This study substantiated Norell's contention that the extracapsular fat pads could easily be identified on the standard roentgenogram of the elbow joint. It further demonstrated the constancy of the soft tissue findings in the normal elbow. The alterations in the soft tissue shadows due to the presence of a traumatic effusion very often were much more obvious than the roentgen findings caused by the minor cortical infractions or subtle fracture that had produced the effusion. Table VII shows a comparison between the present series and Norell's series.

DIFFERENCES IN FINDINGS

The chief differences in the findings in this series of cases and that of Norell were (1) the disappearance of the ventral fat pad in the presence of a visible dorsal fat pad and (2) the failure to see both fat pads in the presence of an intracapsular fracture. Both occurred in a sizable number of cases. Concerning the first finding, two factors were felt to be operating in order to cause disappearance of the ventral fat pad. As was previously mentioned, the ventral fat pad actually is a summation of both radial and coronoid fat pads. When the capsule becomes acutely distended, the alteration in its shape favors a failure of the two shadows to superimpose and thus decreases the spatial illumination gradient. Secondly, the concomitant extracapsular swelling with severe trauma further augments this effect. With regard to the second finding, the disappearance of both ventral and dorsal fat pads in the presence of intracapsular effusion, a decrease in the spatial illumination gradient again was felt to be the primary cause.

SIGNIFICANT CONSIDERATIONS

In a considerable number of cases, the dorsal extracapsular fat pad was not seen in circumstances that warranted its appearance if an intracapsular effusion were the only prerequisite for visualization. Undoubtedly, the explanation for this alteration in the soft tissue findings which appears at variance with Norell's findings, is related to physical and physiologic factors affecting the spatial illumination gradient as discussed by Tuddenham[10] in a recent paper. This seems certain since, when the soft tissue swelling in the extracapsular areas increased and as obvious edema appeared in extensively displaced fractures, the incidence of this type of soft tissue alteration increased markedly. This type of atypical pattern was also seen in ex-

TABLE VII

COMPARISON OF SOFT TISSUE FINDINGS WITH THOSE OF NORELL[7]

Soft Tissue Finding	Soft Tissue Pattern		Norell's 156 Fractures		This Series' 150 Fractures	
			No.	Per Cent	No.	Per Cent
Normal	Ventral +	Dorsal o	30	19	30	20
Evidence of intracapsular effusion	Ventral +	Dorsal +	118	75	71	47
	Ventral o	Dorsal +	o		29	20
Abnormal but without evidence of effusion	Ventral o	Dorsal o	o		20	13
Not evaluated because of overlying plaster	—		8	5	o	

tremely obese patients and in dislocations with marked extracapsular soft tissue edema.

A number of serial roentgenograms demonstrated changes that also helped explain some of the apparent atypical findings on the original roentgenograms. At times, when the original roentgenogram showed a normal soft tissue pattern, a roentgenogram taken several hours or even days later demonstrated the expected or abnormal soft tissue shadows. This is probably due to the fact that at the time the first roentgenogram was taken, a sufficient amount of effusion to displace the dorsal fat pad posteriorly had not yet developed. Conversely, serial roentgenograms also demonstrated a disappearance of the dorsal fat pad in a period of from three to ten days. Thus, if a number of days have passed prior to the examination, normal soft tissue findings may be seen in the presence of an intracapsular fracture.

Another finding not mentioned by Norell was the visualization of a radiolucent shadow on the posterior aspect of the humerus, at a level lower than the usual position of the dorsal pad. While in some cases this may be due to peritendinous fat, because it was most often seen when the elbow was not flexed to a right angle, it very likely represents the extracapsular fat not pressed into the olecranon fossa by the triceps tendon. Hence, care must be exercised to insist on right angle flexion during roentgenography of the elbow. The limits of the capsular attachments must also be considered. A number of elbow fractures are actually extracapsular and, therefore, would not necessarily cause an intracapsular effusion. In these instances, there is a fracture at the elbow joint with no specific abnormal soft tissue finding.

Another factor which seems to have a possible bearing on the incidence of atypical patterns is age. In older patients, and especially males, the shape of the condyle changes and a deepening of the olecranon fossa necessitates a larger intracapsular effusion to produce displacement to the point where visualization would be likely. Considerable distortions of the shape of the distal humerus occurring as the result of old healed fractures were observed in situations where the soft tissue pattern did not conform to the expected pattern. Pathologic deformities of the bone also produced this effect. The best example of this was a case of osteitis fibrosa cystica which so distorted the anatomy of the distal humerus that neither ventral nor dorsal fat pad could be visualized.

A number of widely displaced fractures at the elbow exhibited a normal soft tissue pattern. It is likely that a capsular laceration did not permit an effusion to displace the soft tissue pattern. A normal pattern was often seen in the presence of a severe dislocation. This also may be explained on the basis that a capsular rupture did not allow distention of the capsule, and thus the dorsal extracapsular fat pad was not displaced posteriorly.

CONCLUSIONS

1. The extracapsular fat accumulations at the elbow joint are readily identifiable on the routine roentgenographic examination.

2. Easily identifiable abnormal soft tissue patterns occur in the presence of any type of fracture, ranging from subliminal to severely displaced. Typically, the dorsal extracapsular fat pad becomes dislodged from the olecranon fossa by the intracapsular effusion and is visualized on the lateral roentgenogram. The ventral fat pad remains visible as in a normal roentgenogram.

3. Atypical patterns, not described by Norell,[7] occur relatively frequently and include the disappearance of the ventral fat pad and the failure of the dorsal fat pad to visualize in conditions where an intracapsular effusion must be present. These atypical findings can be interpreted in terms of the following factors: (a) decrease in the spatial illumination gradient because of extracapsular soft tissue swelling, edema or excessive adipose tissue; (b) the impor-

tance of the time interval in the case of less severe trauma; (c) the anatomic necessity for proper positioning during the roentgenographic examination; and (d) alteration in the shape of the condyles secondary to the aging process and residuals of previous fractures and other pathology.

4. An intact capsule must be present for displacement of the dorsal fat pad, explaining the high incidence of abnormal alterations seen in the presence of dislocations.

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UNUSUAL ROENTGEN MANIFESTATIONS OF OSTEOMALACIA*

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OSTEOMALACIA has been defined as "a disorder of bone tissue characterized by a failure of calcium salts to be deposited promptly in the newly formed bone matrix (osteoid)."[1] In growing children there is, in addition, impaired calcification of cartilage at the zone of provisional calcification with a resulting retardation in endochondral ossification. This is the condition known as rickets. The protein matrix of bone and cartilage is not fully calcified because of a low concentration of calcium or phosphorus in the tissue fluids. The alkaline phosphatase, which is usually high in osteomalacia, may be low in the rare cases of hypophosphatasia.

The disease may be caused by (1) impaired absorption of calcium or phosphorus from the gut (vitamin D deficiency; sprue; celiac disease; pancreatic insufficiency; chronic biliary obstruction; infiltration of the mesenteric lymph nodes by carcinomatosis, tuberculosis, or lymphoma; regional enteritis, lymphosarcoma, scleroderma or amyloidosis of the small intestine; small bowel resection; gastroenterostomy); (2) increased excretion of calcium and/or phosphorus by the kidneys (renal tubular dysfunction with or without acidosis but without renal glomerular failure); ureterosigmoidostomy; and (3) hypophosphatasia (decreased alkaline phosphatase activity). Other diseases producing osteomalacia are hypoparathyroidism, renal glomerular failure, and hyperthyroidism.

Biochemical evaluations reveal a normal or low serum calcium, a low or normal serum phosphorus (most commonly a normal serum calcium with a low serum phosphorus) and usually a high serum alkaline phosphatase level. In rare cases of hypophosphatasia, the alkaline phosphatase is low and the serum calcium and phosphorus are normal or may be elevated.

If the disease is sufficiently far advanced to be detected on roentgenograms, the usual appearance is that of a generalized decreased density of the bones; if it is severe there are associated deformities from bending or fractures because of the weakened state of the skeleton. Pseudofractures may be present and are pathognomonic of osteomalacia. These are radiolucent bands of diminished density of the bone cortex and superficially resemble fractures. Pseudofractures are frequently multiple in which case they are almost symmetric. They have certain sites of predilection, such as the axillary border of the scapulae, the ribs, the pubic rami, the medial aspect of the neck of the femur, the iliac bones, the radii, the ulnae, and the tibiae. The cortical defects appear to be produced by erosion of the softened cortex from pulsating vessels in close proximity to the bones.[14]

Many cases of osteomalacia have been described in which the bones appeared to be of normal density roentgenologically, and in some instances pseudofractures have been present. To these latter cases Albright has applied the term "Milkman's syndrome."

OSTEOMALACIA WITH THICK SHORT DENSE BONES AND MULTIPLE ACCESSORY BONES ABOUT THE JOINTS

The following 3 cases are examples of osteomalacia due to familial hyperphosphaturia ("vitamin D resistant rickets") in which the bones were unusually dense and in which there were multiple extra centers of ossification about the joints.

CASE 1. A.F., a forty-four year old male, had a

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chief complaint of intermittent pain in the right knee for one year, and shooting pains in the right hip for one month.

About fifteen years previously, the patient first noticed stiffness and recurrent, mild pain of both shoulders and elbows which was most marked on the right side. The pain in the upper extremities had not been present in recent years but he had limitation of motion in the shoulders. About five years previously, he became aware of limited abduction of the hips. A year later he first experienced low back pain, more pronounced on the right side, which had since been present intermittently. This pain was relieved by weak analgesics.

Physical examination revealed a short, muscular individual who had less strength in his hand shake than his muscular development suggested. He was edentulous having lost his teeth at the age of nineteen years. There were loss of libido with testicular atrophy, prominent breast development over the past five years, and bowed legs.

Laboratory findings showed a serum calcium of 10.5 mg. per 100 cc. and 9.8 mg. with a low serum phosphorus of 2.3 mg. and 1.9 mg., and a markedly elevated alkaline phosphatase of 24.3 Bodansky units. The urinary calcium excretion was normal (35 mg. in twenty-four hours). The urinary phosphorus excretion was 930 mg. in twenty-four hours, which was elevated in view of the intake and the low serum value. Since the creatinine clearance was normal (134 cc. per minute), the patient had a diminished renal tubular reabsorptive capacity for phosphorus (T.R.P. of 74.5 per cent—normal 80 to 90 per cent). The urinary amino acid excretion was normal (alpha amino nitrogen 126 mg. in twenty-four hours).

Roentgen examination. The tubular bones were thick, short, and increased in density, resembling in some respects those of an achondroplastic dwarf. The humeri, radii, and femora were bowed. Rough, irregular bony prominences were present along the lateral aspect of the humeri, the linea aspera of the femora, and the surfaces of the radii, ulnae, tibiae, and fibulae at the point of insertion of the interosseous membranes. The increased density of the bones appeared to be the result of an overabundance of cancellous bone rather than excessive calcification. The cortex in some regions had lost the homogeneous density normally seen in compact bone. Instead, it had some of the characteristics of cancellous bone in which the vertical elements predominated and the trabeculae were packed close together. There was thinning of the cortex in some regions where it was replaced by cancellous bone (Fig. 1, 2, 3 and 4). Numerous small extra centers of ossification were present about many of the joints. These had the general texture of the adjacent bony structures (Fig. 1, 3 and 5). In some regions bony masses, similar to the extra centers of ossification, were attached to the adjacent bone surfaces in the form of exostoses.

The skull was of normal size and shape. The calvarium was thick due to an increase in the size of the diploe and possessed a thin outer table. The vertebral bodies were dense and had

FIG. 1. Case 1. Left hand and wrist. The tubular bones are short, broad, and dense. Small bone fragments are present about the radioulnar joint and between the radioulnar articulation and the carpal bones.

hypertrophic spurs but no evidence of fracture or pressure deformities. The neural arches and their appendages were thick. The clavicles and ribs appeared similar to the long bones of the extremities. The pelvis was dense; the iliac crests were thickened and irregular.

The above chemical findings were in keeping with renal tubular osteomalacia due to hyperphosphaturia, and treatment was initiated with 50,000 units of vitamin D twice daily in September, 1955. Within two months there was marked lessening of all aches and pains, and all joint symptoms except stiffness of the right knee disappeared. The outstanding chemical changes on vitamin D therapy were a fall in the serum alkaline phosphatase towards normal, with little change in either serum calcium or serum phosphorus level. The hyperphosphaturia remained.

A bone biopsy performed by Dr. Joseph Picchi and interpreted by Dr. Jackson Crane was reported as follows: "The cortex of the iliac crest is thick and comprised of rather coarse bone with prominent haversian canals. Subcortical trabecular bone is likewise thick and irregular. The osteocytes are not remarkable. Cement lines are irregular. The trabecular surfaces show no evidence of active osteoblastic or osteoclastic activity. Osteoid seams are not present. The marrow is fatty, with a few scattered islets of hematopoietic cells. Impression: Bone of abnormally coarse trabecular structure."

Fig. 2. Case 1. Right humerus. The shaft is thick and bowed. The bone is dense and the supracondylar ridges are prominent.

Fig. 3. Case 1. Right forearm. The bones are thick and dense. The radius is bent and excessive proliferation of bone has occurred along the upper ulnar margin in the region of attachment of the interosseous ligament. A small bony ossicle is partially superimposed upon the ulnar styloid process.

Case II. C.F., a thirty-eight year old male and brother of A.F., entered H. C. Moffitt Hospital on May 13, 1955. He complained of pain and stiffness in the joints of his arms, fingers, and shoulders which had been present as long as he could remember, becoming progressively worse in the past three years. He had noted in the preceding two years, on several occasions, a generalized aching of his extremities with occasional chills and fever.

The patient had well developed musculature, short extremities, and a normal sized trunk. The

FIG. 4. Case 1. Right tibia and fibula.

legs were moderately bowed and there was some limitation of motion in the arms at the shoulder and in the lower extremities at the hips and ankles. No bone tenderness was elicited. He had a large dome-shaped head. He was edentulous having lost his teeth at an early age. His brother and mother also had poor teeth that had been removed before puberty.

The laboratory findings included a corrected creatinine clearance of 103.8 cc. per minute with a nonprotein nitrogen of 27 mg. per 100 cc. The serum carotene level, vitamin A level, starch and glucose tolerance tests were normal. A phenolsulfonphthalein test showed 37 per cent excretion in fifteen minutes. The serum calcium was 9.2 mg., the serum phosphorus 1.9 mg., and serum alkaline phosphatase 8 Bodansky units. The latter two values, typical of osteomalacia, led to further studies including calcium tolerance and phosphate clearance tests. These showed a normal serum and urine calcium excretion, but an excessive phosphate excretion for the level of serum phosphorus with a low rate of tubular reabsorption of phosphate (T.R.P. 75.3 per cent—normal 80 to 90 per cent). The amino acid excretion in the urine was normal (alpha amino nitrogen 123 mg.).

A bone biopsy was carried out by Doctor Joseph Picchi and interpreted by Doctor Jackson Crane as follows: "The cortex of the iliac crest is comprised of spongy bone which blends imperceptibly with the subjacent medullary trabecular bone. The cortical as well as the medullary bone is comprised of broad, irregular, relatively closely spaced bone trabeculae. Haversian canals are quite prominent throughout the cortex. The bony lamellae are irregular and cement lines are indistinct. There are, throughout, numerous areas of notching associated with the presence of osteoclasts and fibrous type cells. In addition, some of the trabecular surfaces are lined by active appearing osteoblasts subjacent to which are occasional osteoid seams. Impression: Bone with abnormally coarse trabecular pattern and osteomalacia."

Roentgen examination. The roentgen appearance was almost identical to that of Case 1 and will not be described in detail (Fig. 7, 8 and 9). One difference was the shape of the head which in this patient was narrow and long. This ap-

FIG. 5. Case 1. Right hip. The innominate bone is dense and thick. Numerous bone fragments are present about the upper margin of the hip joint.

Fig. 6. Case II. Skull. Dolichocephalic deformity due to premature closure of the sagittal suture.

pears to be the result of premature closure of the sagittal suture (Fig. 6).

Fig. 7. Case II. Left hand and wrist. Bone fragments are present between the ulna and carpal bones. Others are located about the middle proximal interphalangeal joint and the first, second, and third metacarpophalangeal joints. Small exostoses arise from the periarticular surface of many of the bones.

Fig. 8. Case II. Lateral projection of the right elbow. Exostoses arise from the olecranon process of the ulna, and the periarticular regions of the humerus and ulna. Bone fragments are located anterior to the humerus. The trabeculae of the cancellous bone are thick and dense.

On June 10, 1955 treatment was started with vitamin D, 50,000 units once a day; subsequently this was increased to 250,000 units per day. Within two weeks the aches and pains diminished and gradually all symptoms except some joint stiffness disappeared. The outstanding chemical change was a fall in the serum alkaline phosphatase toward normal, while the low serum phosphorus and high urine excretion of phosphorus remained unchanged. On one occasion the urinary calcium became markedly elevated, necessitating reduction in vitamin D dosage to 50,000 units daily, on which dose he is

Fig. 9. Case II. Pelvis and hips. The arrow points to a pseudofracture above the lesser trochanter of the left femur. This is roentgenographic evidence of osteomalacia.

FIG. 10. Case III. Right leg. Rachitic changes are most prominent at the distal end of the femur.

being maintained. The patient is free of symptoms at present. Serial follow-up roentgenograms failed to show any change in the bones.

CASE III. A.M.F., a three year old girl, was the daughter of C.F. Birth and developmental history were normal.

On physical examination the patient weighed 32 pounds and was 36 inches in height. These measurements were approximately in the fiftieth and tenth percentile, respectively, for her age. The head was markedly dolichocephalic; frontal bossae were prominent. The teeth were in good condition. The dorsal thoracic curvature was flattened. The epiphyseal areas of the wrist, ankles, and knees were widened, and the costochondral junctions were palpable. She experienced no pain or stiffness.

Laboratory findings showed a serum calcium of 9.1 mg. per 100 cc., phosphorus 3.1 mg. (low for patient's age), alkaline phosphatase 16.6 Bodansky units, and creatinine 0.36 mg. The urine phosphorus excretion on a diet omitting milk and cheese for four days was 152 mg. in twenty-four hours with a creatinine of 152 mg. The alpha amino nitrogen excretion was normal (27 mg. in twenty-four hours). These findings were considered compatible with a diagnosis of rickets, presumably the result of increased excretion of phosphorus by the renal tubules. The urinary phosphorus, though not excessive, was considered high in view of the low serum phosphate level for a child of three. The diagnosis was further strengthened by the similar chemical pattern in several adult members of her family.

Roentgen examination. The bones appeared to be more dense than those in the average child of this age. The trabeculae were coarse. The epiphyseal cartilages were widened at the ends of the long bones and the zones of provisional calcification were irregularly calcified. These changes were most prominent at the distal ends of the femora (Fig. 10) but were also present at the proximal and distal ends of the tibiae and the distal ends of the radii and ulnae. These abnormalities were typical of rickets.

The skull was long and narrow. The sagittal suture was obliterated. The appearance was that of premature closure of the sagittal suture (Fig. 11).

Treatment was started on May 9, 1956 with 25,000 units of vitamin D per day. The dose was subsequently raised to 50,000 units daily. Whereas the serum levels of calcium and phosphorus remained nearly stable, the alkaline phosphatase at first fell and then rose to a maximum of 20.6 Bodansky units. Follow-up roentgenograms showed signs of healing rickets on March 13, 1957.

The child has continued to grow normally, though at a slow rate. She is fully active and asymptomatic. When last seen, she weighed 40

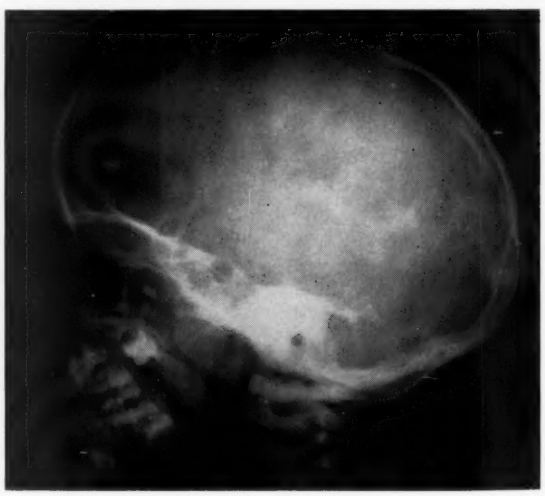

FIG. 11. Case III. Dolichocephalic deformity of the skull due to premature closure of the sagittal suture.

pounds and measured 40¾ inches in height, and was being maintained on 25,000 units of vitamin D daily.

Discussion. The 3 cases are members of a family who suffered from hyperphosphaturia due to impaired reabsorption of phosphate by the kidney tubules. No glycosuria or amino-aciduria has been observed in any of them. The increased excretion of phosphate in the urine is presumably responsible for a low inorganic phosphorus concentration in the serum, and subsequent osteomalacia.

The hereditary forms of rickets and osteomalacia have been described in detail by Dent and Harris[6] and by Winters *et al.*[15] Our cases would fit in with Dent's subgroup described as "milder rickets presenting in childhood which closely mimics dietary deficiency of vitamin D and requires moderately large doses of vitamin D to produce healing." These patients may have no obvious abnormality until the age of three or four years, or later. Then only a lack of stature may be noted until adult life, when signs and symptoms appear. The disease is usually of a similar severity in all involved members of a family.

The adult patients resembled achondroplastic dwarfs because of their short stature and disproportionate length of their extremities and spine. Their muscles were very well developed and the long bones were thick and dense. Indeed, the original roentgen diagnosis of the adults was achondroplasia. The diagnosis of chondrodystrophy was suggested in 3 of 5 patients with vitamin D resistant rickets described by Holt.[12] The difficulty in differentiating between achondroplasia and hyperphosphaturia on roentgenograms is greater in adults after the epiphyses have closed and the characteristic signs of rickets are not present.

The bones of the 3 patients presented appeared to be more dense than normal on roentgenograms. This was due to the increased thickness of the bones containing an abundance of trabeculae that were only slightly demineralized. The cortex lacked the usual homogeneous density of compact bone. Engfeldt, Zetterström, and Winberg[9] examined biopsies of costochondral junctions in 3 cases and of the tibia in 1 case of hyperphosphaturia by means of the conventional microscope, microradiography, and polarized light. They found in the costochondral junctions uncalcified osteoid tissue in large amounts which was similar to that seen in vitamin deficiency rickets. In many areas, irregular bone trabeculae replaced the outer lamellae and compact bone. In the tibia of a patient who had been treated but in whom improvement was not complete were broad trabeculae separated by wide areas of bone marrow. The osteoid was scanty and osteoblastic borders and resorption cavities were frequently noted. The distribution of mineral salts as seen on microradiograms varied widely. There was an occasional accentuation of fine lamellar structure and some highly mineralized trabecular areas. Other areas showed evidence of bone destruction and contained broad zones of poorly mineralized tissue. The over-all mineralization was almost normal. The compact bone presented a mosaic structure resembling Paget's disease.

Another interesting finding on the roentgenograms of the 2 adult patients was the presence of multiple small extra ossification centers in most of the joints. In some regions there were associated degenerative changes of the adjacent articular cartilage and subchondral bone. This was probably the cause of limitation of joint motion. Almost identical extra bone centers of ossification were found by Finby and Bearn[10] in patients with hyperphosphaturia and osteomalacia associated with Wilson's disease. The presence of these unusual bone fragments was seen in 9 of 20 patients studied. The similarity between their illustrated cases of lesions of the wrists and elbows and 2 presented in this report is most striking. The lesions were most prominent in the wrists just distal to the ulna, the anterior aspect of the elbow joint, and about the hips. They believed that the bone

fragmentation, osteoarthritis, and cartilage degeneration in Wilson's disease were not related to the disturbed renal function and were possibly directly due to the abnormal copper metabolism present in these patients. However, the almost identical joint abnormalities seen in our patients would indicate that these highly characteristic lesions are indeed the result of the kidney disturbance and associated osteomalacia. We have seen these lesions in several other patients with hyperphosphaturia and osteomalacia. They are probably another manifestation of the irregular endochondral ossification occurring at the ends of the bones. These patients also have prominent exostoses in the regions of tendinous and ligamentous insertions. These are most likely due to stress applied to bone that has become weakened by depleted mineral stores and is excessively active.[2]

The unusual shape of the skull of Cases II and III is the result of early closure of the sagittal suture. Craniostenosis has been described many times in association with osteomalacia due to renal tubular dysfunction.[3,13]

OSTEOMALACIA WITH ASSOCIATED SUBPERIOSTEAL CORTICAL RESORPTION AND LOCALIZED DESTRUCTIVE LESIONS

CASE IV. M.M. This case has been described previously, but the entire diagnosis had not been established at that time.[14]

A sixty-two year old white female entered the University of California Hospital on August 5, 1952 with a history of progressive backache. Two years before admission she began to have pain in her back, radiating anteriorly. She also had some pain in her right hip, aggravated by walking. Injections of cortisone for about three months had caused an improvement in appetite but did not alleviate the pain.

Physical examination showed a small, thin woman in moderate distress, unable to move about freely. The important findings were: chest markedly deformed, with a dorsal kyphoscoliosis; rib cage unduly springy, with tenderness along the ribs in the left axillary line; distal end of the right clavicle deformed; heart enlarged, with a Grade 2 systolic precordial murmur. There was some tenderness in the region of the costovertebral angles and swelling of the interphalangeal joints of the hands.

Laboratory findings showed a serum calcium 8.5 mg. per 100 cc., phosphorus 3.0 mg., alkaline phosphatase 78 Bodansky units, protein 6.5 gm., albumin 4.0 gm., chlorides 100 mEq. per liter, carbon dioxide 22.6 mEq. per liter, sodium 138 mEq. per liter, potassium 3.3 mEq. per liter. Sulkowitch tests were consistently negative. The twenty-four hour urine test contained 20 mg. of calcium. Glucose tolerance tests showed fasting blood sugar to be 121 mg. per 100 cc., the half-hour figure was 210, one hour 337, two hours 269. Plasma carotene was 0. Stool fat at seventy-two hours: weight 341.8 gm. quantity, 3.4 per cent wet weight, 17 per cent dry weight; total fat: 11.6 gm. total wet weight. Vitamin A tolerance, starch tolerance, and gelatin tolerance tests were all borderline.

Roentgen examination. The entire skeleton was markedly decalcified. In addition, there were numerous cortical defects which in some instances were almost bilaterally symmetric. These pseudofractures were most prominent in the distal thirds of the clavicles, bases of the acromion processes of the scapulae, posterior and lateral surfaces of the ulnae, and the superior and inferior pubic rami (Fig. 12). These findings indicated the presence of osteomalacia.

Superimposed upon the changes due to osteomalacia were the following roentgen abnormalities, indicating that the patient was also suffering from hyperparathyroidism: destructive lesions of various sizes in the inferior ramus of the pubis on the left side, the head of the right second metacarpal bone, the distal phalanges of the right second and fourth fingers, the middle phalanx of the left second finger, the distal phalanx of the left fifth finger, and the anterior aspect of the right tibia (Fig. 13 and 14). The lesions with the exception of the tibia showed no evidence of bone production around their margins. The lesion in the tibia was very extensive and presented a saucer-like defect along the anterior surface of the proximal half. This lesion was surrounded by a margin of bone of greater density than the surrounding bone. Other signs of hyperparathyroidism were: minimal subperiosteal resorption of cortical bone which was most obvious in the middle phalanges of the hands; an absence of the lamina dura of the jaws (Fig. 15); and a mottled appearance of the calvarium of the skull (Fig. 16).

A roentgenogram of the abdomen revealed

clacification within the body and tail of the pancreas.

A biopsy of the lesion of the right tibia and of the surrounding bone was reported as follows: "The normal dense cortical bone of the tibia has been transformed into markedly thickened spongy bone. This bone is comprised of thick, irregular, closely spaced trabeculae between which is a cellular fibrous tissue of relatively poor collagen content. Most of the trabeculae are, in great part, lined by a single layer of active osteoblasts. Occasionally, a subjacent osteoid seam can be identified. Many of the trabeculae are notched in an irregular manner, indicating bone resorption. In most of the notches, one or more osteoclasts can be identified. There has been a degree of subperiosteal bone resportion and replacement with cellular fibrous tissue. This zone blends perceptibly into the subjacent coarse spongy bone of the cortex. Impression: Severe osteomalacia characterized by the transformation of the cortex into coarse spongy bone with excessive resorption of bone and deposition of new bone."

On August 23, 1952 the patient was started on calcium lactate powder, 10 gm. per day; vitamin D (Condol) 50,000 units per day; high carbohydrate, low fat, low protein, and high calcium diet; multivitamin tablets; and pancreatin 3.0 gm. three times daily. On this regimen, the urinary calcium had become weakly positive by September 8, 1952. In addition, calcium chloride was given in doses of 6 gm. per day, starting on September 17, 1952.

Fig. 12. Case IV. Abdomen and pelvis. The superior and inferior pubic rami are the sites of pseudofractures indicating that the patient has osteomalacia. The bones are generally demineralized. Diffuse calcification of the pancreas is demonstrated.

Fig. 13. Case IV. Right hand. Destructive lesions are present in the second and fourth distal phalanges, the head of the second metacarpal and the distal end of the radius. The tufts of the distal phalanges are eroded and there is subperiosteal resorption of the cortex of the middle phalanges which is not demonstrated on the reproduction. These are signs of hyperparathyroidism.

A skeletal survey on September 30, 1952 demonstrated a marked repair of the localized destructive lesions, pseudofractures, and subperiosteal cortical erosion. The entire skeleton was still decalcified but this was less than on the previous examination.

There was marked and rapid improvement of

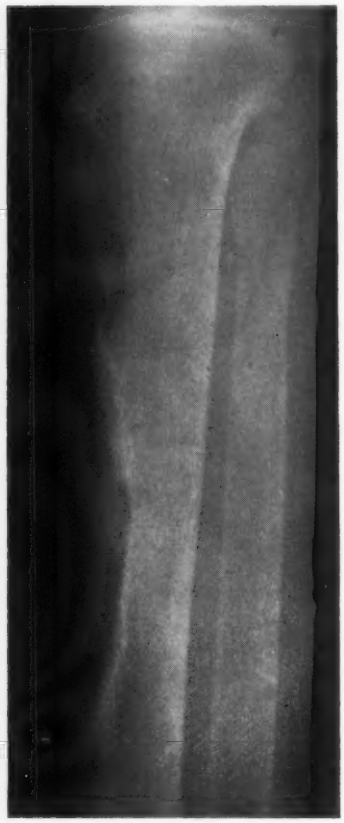

FIG. 14. Case IV. Large defect of the anterior aspect of the right tibia due to hyperparathyroidism.

the patient's general outlook, bone pain, and mobility. Laboratory reports on December 19, 1952 showed a return of all the chemical values to normal. Roentgenograms revealed a change in all of the bony defects which had now become more dense than the surrounding skeletal structures. The previously observed hypertension, unfortunately, continued to mount, and the patient developed a cerebral hemorrhage and expired.

The cause of death was cerebral infarction, secondary to hypertensive cardiovascular disease. In addition, there was far advanced pancreatic atrophy and fibrosis associated with pancreatic ductal lithiasis. Bone tissues of the ribs, vertebrae, pelvis, and femur were examined. Grossly, these bones presented with relatively thin cortices and delicate medullary trabeculations. The marrow was, in general, fatty, although there were focal areas of hematopoietic-forming marrow in the vertebrae. Microscopically, the cortices were thin but regular, with normal appearing haversian systems and lamellar bone formation. The subjacent trabeculae were thin, delicate, and relatively widespaced. There was no evidence of excessive osteoblastic or osteoclastic activity. No osteoid seams were identified. The impression was that at this time there was a degree of osteoporosis with no evidence of osteomalacia. Three of the four parathyroid glands were identified. These were of normal size and showed the usual distribution of chief cells and oxyphil cells arranged in cords between which were interspersed groups of fat cells. At the time of autopsy the bones were found to be quite firm.

Discussion. This patient had osteomalacia, presumably due to chronic pancreatitis. The low serum calcium and high alkaline phosphatase without calcuria provided chemical evidence of impaired absorption of calcium from the small intestine. The

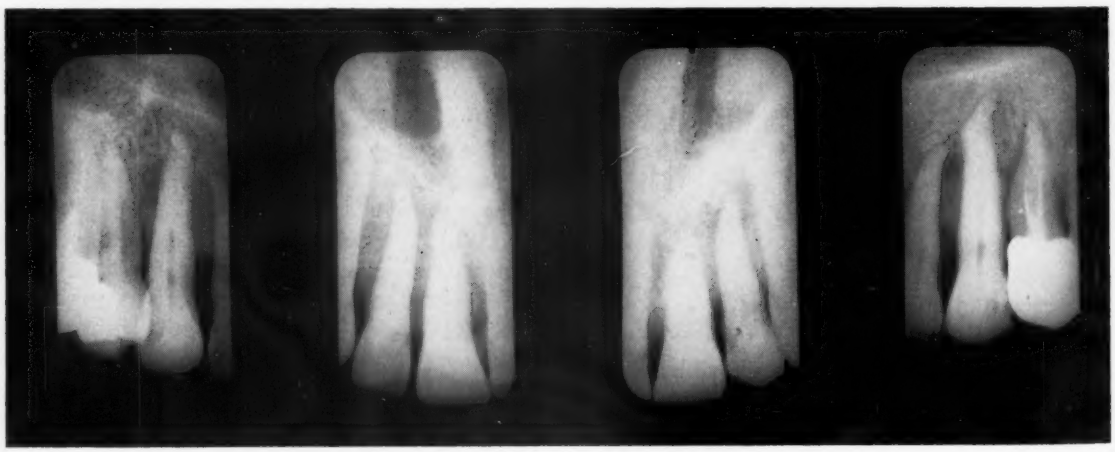

FIG. 15. Case IV. Dental roentgenograms. The lamina dura is absent.

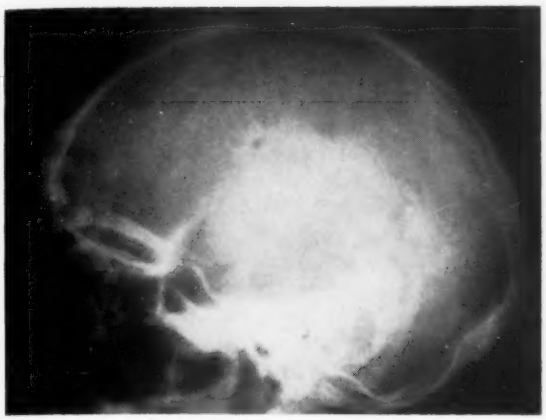

Fig. 16. Case IV. Mottled appearance of the calvarium.

roentgenologic evidence for osteomalacia was the multiple pseudofractures. In addition, however, this patient exhibited roentgen signs which we consider specific for hyperparathyroidism. These were subperiosteal cortical erosion, absence of the lamina dura of the jaws, localized destructive lesions, and a mottling of the calvarium of the skull.[5,7] The blood calcium and phosphorus returned to normal after adequate treatment for steatorrhea and osteomalacia. The roentgen signs of osteomalacia and hyperparathyroidism also reverted toward normal with continued treatment. At the time of autopsy the parathyroid glands were normal.

Two cases of the association of hyperparathyroidism and steatorrhea have been reported by Davies, Dent, and Willcox.[4] In their cases, as in ours, the hyperparathyroidism was assumed to be present on the basis of the roentgen findings. In one of their cases, treatment with vitamin D led to a remission of the bone pains and of the roentgenologic signs of osteomalacia and hyperparathyroidism. In their second patient, two parathyroid adenomata were removed surgically after which symptoms were relieved and the roentgenologic signs returned toward normal.

It thus appears that some patients with osteomalacia develop a secondary hyperparathyroidism presumably as a result of their low serum calcium.[4,8,11] In the absence of any direct means of parathyroid hormone assay, a roentgen examination is the only simple means of determining hyperparathyroidism in the presence of osteomalacia. The surprising thing is not that the two diseases may be associated, but that the association is not more commonly found. It is known that primary hyperparathyroidism occurs in many patients in whom there is no roentgen evidence of bone involvement, although bone biopsies demonstrate osteitis fibrosa. Hyperplasia of the parathyroids with secondary hyperparathyroidism probably occurs in many patients with steatorrhea in whom the changes in bone are not far enough advanced to be detected by the ordinary roentgen methods.

SUMMARY

The case histories of 3 members of a family with osteomalacia due to hyperphosphaturia are presented. Roentgenograms revealed the bones to be denser than normal. Characteristic extra centers of ossification in some of the joints and excessive proliferation of cortical bone at the insertion of tendons and ligaments were noted in the 2 adult patients. Two patients had premature closure of the sagittal suture of the skull.

A case of osteomalacia due to chronic pancreatitis with typical changes of demineralization and pseudofractures is also described. In addition, this patient had roentgen evidence of secondary hyperparathyroidism manifested by subperiosteal cortical resorption, absence of the lamina dura of the jaws, mottling of the calvarium, and localized destructive lesions of the bones.

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Vol. 82, No. 5

ROENTGENOGRAPHIC FINDINGS IN THE COMPLICATIONS OF DIABETES MELLITUS

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FEW diseases definitely associated with one organ and one metabolic function have such widespread concomitant pathologic changes as does diabetes mellitus. The protean nature of this disorder has been appreciated clinically for many years and there have been many reports in the radiologic literature concerning certain aspects of these pathologic changes, which are demonstrated roentgenographically. Indeed, certain roentgen changes are almost pathognomonic and other changes occur with such frequency as to be highly suggestive of the disease.

Although the literature contains a number of communications dealing with specific lesions found in diabetes mellitus, no comprehensive review of the roentgenographic findings in all systems of the body has been presented. It is the purpose of this paper to review the roentgen changes encountered in diabetes mellitus as well as to point out several findings which have not previously been described.

MATERIAL

The clinical records of 672 cases of diabetes mellitus admitted to the Protestant Deaconess Hospital over a three year period have been reviewed. There were 177 cases examined roentgenographically and on these cases a total of 547 different examinations have been performed. From these examinations most of the examples of roentgenographically evident lesions encountered in diabetes mellitus were obtained.

GENITOURINARY SYSTEM

The genitourinary system is the frequent site of a number of complications in diabetes mellitus and there probably has been more written on this phase of the roentgenographic findings than on any other phase.[8,10,12,14,15,16,17,20,21,27]

Necrotizing renal papillitis is found only in cases of diabetes mellitus or in obstructive uropathy with secondary infection; therefore, in the absence of obstructive uropathy, it should suggest the diagnosis of diabetes mellitus. Initially, there is calyceal haziness which is followed by sinus tract formation and eventually a complete ring shadow. The papilla becomes extruded, leaving a club-like fossa (Fig. 1). The extruded papilla may be seen as a negative shadow in the renal pelvis (Fig. 2). The end stage of healing of this lesion is sometimes incrustation by calcium salts, thereby producing calcific ring shadows apparent on plain roentgenograms (Fig. 3). In the sinus formation stage, confusion may arise with pyelocanalicular backflow. While pyelocanalicular backflow occurs in normal functioning kidneys, an acutely ill patient with progressive renal failure indicates necrotizing renal papillitis.

Gas-forming infection in the perirenal tissues is the next common complication. Olson[20] in a recent communication gave an excellent review of this subject. *Escherichia coli* is nearly always the causative agent. Multiple small bubbles of gas in the peri-

FIG. 1. The upper pole calyces on the right have coalesced and present as a trilobate common calyx. The remainder of the collecting system appears to be normal. This probably represents the club-like fossa stage of renal papillary necrosis.

Fig. 2. There are multiple blunt calyces present. In the center of the renal pelvis is a radiolucent shadow which may well represent an extruded papilla in an advanced stage of renal papillary necrosis. These findings were present in a known diabetic in renal failure.

renal area are quite characteristic in appearance and the mere presence of a perirenal gas-forming infection should suggest the possibility of diabetes mellitus (Fig. 4).

Gas-forming infections of the bladder are also a fairly frequent occurrence.[12] Cystitis emphysematosa is recognized by a halo of gas about the bladder shadow (Fig. 5). The condition is transient but almost invariably occurs in diabetics. The transient nature may be the reason for its infrequent observation. The condition has been produced experimentally in animals by the intravenous injection of glucose and simultaneous infection of the bladder with *Escherichia coli*. The blebs are recognizable on cystoscopic examination as well as roentgenographically. Primary pneumaturia is also found but usually as a complication of neurogenic bladder secondary to diabetic neuropathy. In these cases the examination for the urine sugar may be negative due to utilization of the sugar by the *Escherichia coli*.[10]

Diabetes mellitus may sometimes be the underlying disease in neurogenic bladder dysfunction,[1] the so-called "cord bladder," manifested roentgenographically by a large bladder size and reflux of opaque material into the ureters, which are also large in caliber. As previously stated, the high urine concentration of sugar combined with urinary stasis and *Escherichia coli* infection may lead to pneumaturia. Calcification of the vas deferens and seminal vesicles has been described on the basis of pathologic observations,[28] but this finding was not encountered roentgenographically in the present series of cases.

Fig. 3. Club-shaped upper and lower pole calyces visualized by retrograde pyelography. The parallel dense shadows in the lower pole probably represent calcium incrustations in a destroyed major calyx. This is believed to be the end stage of renal papillary necrosis.

Fig. 4. Multiple small gas bubbles in the perirenal tissues with indistinct kidney mass outline. The minor calyces are blunt. The infection was drained surgically and proved to be due to *Escherichia coli*.

Adenocarcinoma of the corpus uteri may also be related to diabetes mellitus in that its frequency of occurrence is increased. This complication may prove of concern to the radiologist as a therapeutic problem.

GASTROINTESTINAL SYSTEM

In a review of 72 cases of diabetes mellitus with dysphagia, 10 examples of ripple esophagus were found (Fig. 6). The youngest patient in whom it occurred was forty-nine years of age. The ripple esophagus is generally considered to be a neuromuscular dysfunction, found most frequently in elderly people. It is presumed that this finding in a diabetic reflects nerve degeneration. Conversely, when a ripple esophagus is encountered in a relatively young person, the possibility of diabetes mellitus as a cause for the dysfunction should be considered.

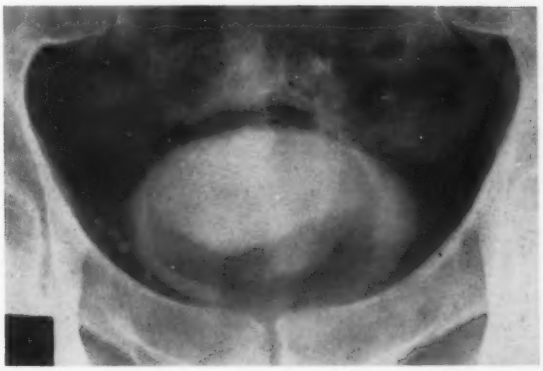

Fig. 5. The halo of radiolucency around the bladder shadow represents multiple small bubbles of gas in the bladder wall. The appearance is quite striking and almost invariably the result of *Escherichia coli* infection.

Acute dilatation of the stomach is a frequent finding in diabetic acidosis. An example of this is shown in Figure 7. A survey roentgenogram of the abdomen on a patient admitted in acidosis with abdominal

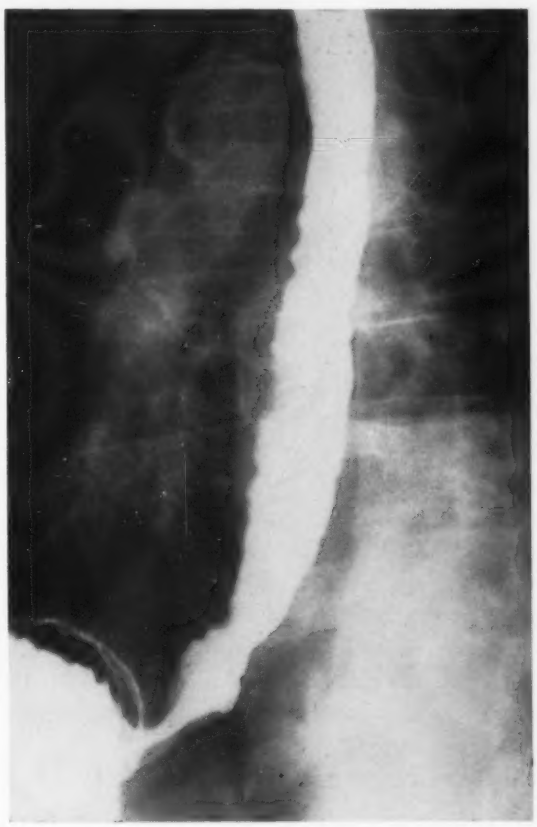

Fig. 6. Ripple configuration of the esophagus in a forty-nine year old known diabetic with dysphagia.

Fig. 7. Acute dilatation of the stomach in a young diabetic admitted in coma with a blood sugar of 520 mg. per cent. The acute dilatation subsides with control of the diabetes.

distention revealed marked gastric dilatation which should not be confused with acute intra-abdominal disease. The acute dilatation promptly returns to normal with control of the diabetes. Diarrhea likewise occurs rather frequently in diabetics, being due to hypermotility of the small intestine. Diabetic nocturnal diarrhea was investigated in 40 cases by Sheridan and Bailey.[26] They performed upper gastrointestinal series in 17 patients and all had normal findings; 28 cases had colon examinations with the only positive finding being spasticity in 6 cases. On the other hand, Hodges *et al.*,[13] in a study of the small intestine in various neurologic diseases, showed examples of delayed gastric emptying, prolonged small bowel transit time, considerable variability in small bowel lumen width and displacement of small bowel loops by a distended bladder in cases of known diabetic neuropathy. The correlation of clinical and roentgenographic findings was vague and, even with control of the diabetes, the roentgen findings would frequently persist. In the present group of cases, the upper gastrointestinal tract was studied in 71; no abnormality, either functional or anatomic, was observed in a single instance. The colon was also studied roentgenographically in 77 cases and, except for 12 cases of mild diverticulosis, no abnormality was found.

Of the diabetic abnormalities of the pancreas which may produce roentgen findings, pancreatitis, either acute or chronic, is the most common. In the acute stage, there may be evidence of multiple small bubbles of gas in the region of the pancreas. This gas-forming infection is almost invariably a complication of diabetes mellitus. The calcific stage of pancreatitis may also be evident roentgenographically. McGeorge and co-workers,[18] in a recent paper reporting 18 cases of diffuse calcification of the pancreas, found that 10 had positive urine sugars and, of the entire group, 6 proved to have diabetes mellitus. An example of the calcific stage of pancreatitis is shown in Figure 8. This patient also had multiple radiolucent gallstones, which might bear a cause and effect relationship to the pancreatitis. Sporadic reports would indicate that cysts or pseudocysts of the pancreas may also be associated with diabetes and may be another manifestation of chronic pancreatitis. The wall of the cyst may be

Fig. 8. Multiple amorphous calcifications in the entire pancreas. The gallbladder is functioning and contains multiple nonopaque stones.

calcified and identified on roentgenograms.[23] Furthermore, it is of interest that the incidence of carcinoma of the pancreas is strikingly increased in diabetes mellitus. Warren and LeCompte[28] state that adenocarcinomas of the pancreas constitute from 2.5 to 4.8 per cent of all carcinomas in nondiabetic individuals, while they constitute 14 per cent of all carcinomas in the diabetic. The usual roentgenographic signs help to establish the diagnosis of carcinoma of the pancreas.

Gas infection of the gallbladder is also noted in diabetes mellitus. *Clostridium welchii* or *Escherichia coli* is the usual offending organism. The infection may cause emphysematous changes in the wall of the gallbladder (Fig. 9) or may fill the gallbladder itself with gas (Fig. 10). The subject of incidence of cholelithiasis and cholesterosis has been debated at great length. Warren and LeCompte[28] found an incidence of 31 per cent gallstones in 453 diabetics and of only 21 per cent in 500 nondiabetics. Feldman and Feldman,[9] on the other hand, found an incidence of 22.7 per cent in 1,319 autopsies, with an incidence of 24.8 per cent in the 137 cases of diabetes mellitus. The same authors found an incidence of 9 per cent of cholesterosis in diabetics and 12.5 per cent in all cases. In the present series a number of cases had gallstones demonstrated on roentgenograms. The types of stones varied between multiple small cholesterol stones, large single cholesterol stones with calcific incrustation, multiple laminated calcific stones and pigment and calcium stones. There did not seem to be a predilection for one roentgenographic type of stone. Fifty cases in this series had cholecystographic examinations and in 19 the gallbladder was found to be abnormal, presenting nonvisualization, gallstones or cholesterosis.

Fig. 9. Emphysematous changes in the wall of the gallbladder in a diabetic with an acute abdomen and palpable right upper quadrant mass. The roentgenographic appearance is quite striking and almost pathognomonic of diabetes mellitus. (Courtesy of Dr. R. J. Nichols, Vincennes, Indiana.)

Fig. 10. Gallbladder containing gas in a diabetic admitted with fever and right upper quadrant pain. A gas-fissured gallstone is noted medial to the gallbladder. At surgery the gallstone was found in the cystic duct.

SKELETAL SYSTEM

The changes in the skeletal system secondary to diabetes mellitus are ascribed to three causes; *i.e.*, neuropathy, avascular necrosis and osteomyelitis. At times, it is difficult to determine whether a given bone

Fig. 11. The first and second lumbar vertebrae are involved in an atrophic process with fragmentation. There also are found hypertrophic spurs laterally, and the over-all density of bone is increased. These joint changes were asymptomatic in a patient with diabetes mellitus of long duration.

lesion is due to one of these causes or to a combination of several of them. It requires a close correlation of history, physical findings and laboratory data to interpret properly the skeletal roentgenographic changes. Pugh[24] states that, although infection and inflammation explain most of these so-called neurotrophic lesions of the bones of the feet, one cannot deny that unusual lesions of bone are encountered sometimes when there are neurotrophic disturbances without inflammation or infection being present.

The so-called diabetic Charcot joints have received the most attention in the literature.[1–5,22,30] The roentgenographic appearance is identical with that of the Charcot joints of other etiologies; thus, there is evidence of fragmentation, eburnation, absorption of articular surfaces, loose bodies in the joint and calcific deposits in the synovial membrane. The ankle is by far the most frequently involved joint. The tarsal and metatarsal articulations are probably the second most frequent sites. Sporadic case reports of involvement of other joints have appeared. In our files we have a typical Charcot spine which was encountered in a known diabetic. There was no clinical or laboratory evidence of luetic infection (Fig. 11).

A far more frequent occurrence is the presence of avascular necrosis. The earliest roentgenographic finding in this condition is demineralization of a spotty variety which is frequently followed by resorption of the tufted end of the terminal phalanges. By the time the avascularity is of such a degree as to produce resorption of bone, frank gangrene is present in the soft tissues with secondary infection occurring to a greater or lesser degree. The osteomyelitis which follows is frequently accompanied by a gas-forming infection in the adjacent soft tissues (Fig. 12). The amount of periosteal reaction to the bone infection is usually less than is seen in osteomyelitis, and the process is primarily a destructive one.

As previously stated, it is often difficult to determine the etiology in a given case. For example, the roentgenogram of the thumb in Figure 13 shows a destructive and fragmented area adjacent to the interphalangeal joint. This occurred in a laborer with diabetic neuritis, manifested by loss of pain and temperature sensation in the upper extremities, who had had a puncture wound two weeks prior to the roentgenographic examination. The neuropathy was evident clinically and incision and drainage revealed frank pus in the area. The lesion, no doubt, represented a septic arthritis in an area where previous, repeated trauma had produced fragmentation. When vascular calcification is present, it helps to establish the cause of the bone abnormality as avascularity. Likewise, the presence of gas bubbles in the soft tissue points to in-

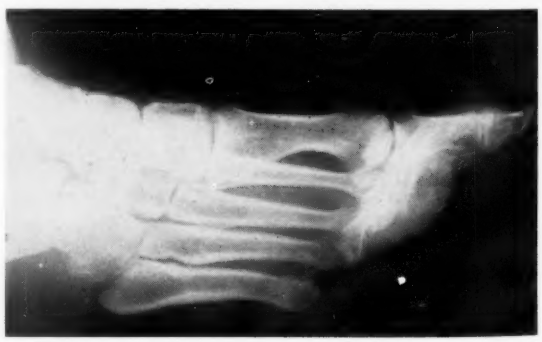

Fig. 12. Gas-forming infection in an area of gangrene in a patient with long standing diabetes mellitus.

Fig. 13. There is fragmentation and demineralization of bone at the interphalangeal joint of the thumb. This patient had a diabetic neuropathy with loss of pain and temperature sensation and sustained a puncture wound two weeks prior to the examination. On incision and drainage staphylococcic pus was found.

fection as playing a causative role. The presence of fragmentation and eburnation of bone should lead one to suspect a neuropathy, although by the time the neuropathy is of such proportions as to produce a Charcot joint, it is usually quite evident clinically.

NERVOUS SYSTEM

Probably the most common abnormality of the nervous system in diabetes mellitus is the so-called diabetic neuropathy.[7,11,29] Estimates as to frequency have varied from 4 to 66 per cent. The etiology of the neuropathy is obscure but the theory with the most proponents is that it is due to arteriosclerotic changes in the vasa nervorum.

The roentgenographic findings are dependent upon which organ is supplied by the affected nerve. Charot joints, tabes diabetica, functional abnormalities of the esophagus, cord bladder, etc., are manifestations of neuropathy that are evident roentgenographically as abnormalities of the organ supplied by the affected nerve. While the changes are in no way characteristic of diabetes mellitus, the presence of neuropathic changes in the various organs should be kept in mind by the clinician in charge of the case, and the radiologist should be cognizant of diabetes mellitus as an etiologic agent.

Acromegaly is a disease due to eosinophilic adenoma of the pituitary in which diabetes mellitus is sometimes found.[6] The changes of acromegaly are manifest roentgenographically, primarily as bone abnormalities, and the radiologist may be called upon to determine the presence or absence of acromegaly in a known diabetic.

CARDIOVASCULAR SYSTEM

The most frequent cardiovascular cause for hospitalization in our series of cases of diabetes mellitus was coronary thrombosis. The changes in the chest roentgenograms were usually minimal and in no way typical of the diabetes. In a number of cases hypertension accompanied the diabetes and led to large hearts with a configuration suggesting a large left ventricle. It is agreed that pathologically the arteriosclerotic changes observed in diabetes are not characteristic of the disease.[19,25] However, Warren and LeCompte[28] noted that intimal thickening and deposition of cholesterol crystals are found in the elastic-vessels and that calcification of the media is seen primarily in the muscular vessels. This deposition of calcium in the peripheral muscular vessels instead of the large elastic vessels is also noted roentgenographically. It is a fairly frequent occurrence to observe in the thirty to fifty year age group extensive calcification of the vessels of the feet with little or no calcific deposits in the aorta. In the older age group the calcification of the aorta is noted with equal frequency and probably can be attributed to the advancing years rather than the altered arteriosclerotic changes of diabetes mellitus.

FIG. 14. Marked calcific degeneration in the pedal vessels of a thirty-nine year old male.

The extensive calcification of the pedal vessels (Fig. 14) is seldom observed in diseases other than diabetes mellitus in patients under fifty years of age. Furthermore, the disparity in calcification of the pedal vessels in relation to calcification of the aorta in this age group is seldom seen in diseases other than diabetes mellitus.

SKIN

Pathologically, there may be amorphous deposits of calcium in the skin and subcutaneous tissue in cases of diabetes mellitus. The deposits lie in the subcutaneous fat as well as in the corium. These deposits have not been recognized roentgenographically in our series.

LUNGS

Two pulmonary abnormalities are encountered in diabetes mellitus with increased frequency. The one abnormality which has been considered as having an increased incidence is hyaline membrane disease. Infants of diabetic mothers have an incidence of 16 per cent of hyaline membrane disease. The cause of this increased incidence is not apparent and the roentgenographic findings of this abnormality in no way differ from those of hyaline membrane disease encountered in infants of nondiabetic mothers.

Pulmonary tuberculosis has in the past been quite common in diabetics and at one time accounted for nearly half of the deaths. There has been a marked decrease in the incidence of this complication, although the decrease has not been as rapid as the decrease of tuberculosis in the general population.

SUMMARY

While the diagnosis of diabetes mellitus is not usually incumbent upon the radiologist, his role in detecting the complications of diabetes mellitus cannot be overemphasized. The complications which are manifest roentgenographically are sometimes so characteristic or occur with such frequency that they are almost pathognomonic of the disease.

Perirenal gas-forming infection, cystitis emphysematosa, and primary pneumaturia are seldom seen in patients who are not diabetics. Bladder paralysis is sometimes observed as a manifestation of diabetic neuropathy and renal papillary necrosis is also encountered. The increased frequency of occurrence of adenocarcinoma of the corpus uteri has long been known and usually only comes under the observation of the therapeutic radiologist.

The presence of a ripple or corkscrew esophagus is believed to be a neuropathic manifestation of diabetes mellitus. This finding has not been previously described in relation to this disease.

Acute dilatation of the stomach is seen in diabetic coma and this condition regresses with control of the disease. There is some disagreement as to whether there are roentgenographic findings in the small bowel or colon when associated gastrointestinal symptoms are present. In our limited series of cases no significant abnormalities were observed.

Acute gas-forming infection of the pancreas is seldom seen in conditions other than diabetes mellitus. Diffuse calcification of the pancreas is also frequently accompanied by diabetes mellitus. Likewise, it should be pointed out that the incidence of carcinoma of the pancreas is increased in this disease.

Gas formation in the wall of the gallbladder and collections of gas in the lumen of the gallbladder are observed. The type, number and incidence of gallstones is apparently unrelated to the disease. One would expect an increased number of cholesterol stones in view of the altered cholesterol metabolism but such, apparently, is not the case.

Changes in the skeletal system are ascribed to neuropathy, avascular necrosis and infection. While none of these changes are absolutely characteristic, diabetes mellitus should be recognized as an etiologic agent in neuropathic joints. Similarly, the premature calcific degeneration in peripheral blood vessels leading to atrophic bone changes is noted with such frequency in diabetics that the roentgenographic appearance may be considered quite characteristic. Osteomyelitic infections which are gas forming should always suggest diabetes mellitus.

The pathologic processes involved in vascular degenerative changes are identical to those found in the normal aging process, although accelerated. However, in diabetics the degree of calcification in the distal peripheral vessels, *e.g.*, pedal vessels, is often out of proportion to the calcific degeneration of the large elastic vessels, as for example, the aorta.

The roentgenographic findings of diabetic neuropathy are reflected in the organ or part supplied by the affected nerve. The changes may involve somatic or autonomic nerves and may consist of sensory or motor alterations. To recognize these functional alterations is of paramount importance for proper management of the diabetic patient.

Diabetes mellitus is not often regarded as a disease which requires roentgenographic confirmation. Its complications, however, can frequently be diagnosed by roentgenologic means.

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PLACENTOGRAPHY WITH DUO- AND TRI-SPEED INTENSIFYING SCREENS*

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DIRECT placentography is the demonstration of the actual placental soft tissue density as a marked widening or bulging of the uterine wall. Since the placenta is located in most cases on the anterior or posterior uterine walls,[2] with the posterior slightly more frequent, the lateral roentgenogram of the abdomen is the best view. The marked differences in thickness and density of the anterior and posterior portions of the abdomen with the patient in the lateral position has been a technical problem. With routine technique it is impossible on a single roentgenogram to obtain proper exposures for both anterior and posterior portions of the abdomen. Two completely different exposures are necessary in order to visualize both anterior and posterior walls at once. An attempt to solve this problem has been the use of filters[1,2,4] (plastic, aluminum, barium paste), wedge-shaped or curved, placed in front of the roentgenographic tube to equalize exposure. Since the filtration is usually fixed and the patients vary considerably in thickness, the results are not uniform nor predictable.

Since February, 1956, when this study was first begun, a new technique for single film lateral placentography has been developed. By the use of combinations of portions of intensifying screens of different speeds in one cassette, the differences in density and thickness of the anterior and posterior portions are provided for. The slower speed screen is for the thinner anterior portion, the faster speed for the thicker posterior portion.

Seven types of intensifying screens were supplied and utilized in various combinations as to relative speed, the number of types in one cassette, and the relative amount of space in the cassette to be occupied by the different types. The specific screens with their manufacturer, full descriptive names, symbols or types as designated by the manufacturer, exposure factors, and relative speed ratio are listed in Table 1.

The first combination used was two 7 inch×17 inch screens (Radelin T and HR), ratio 2:1. This was not satisfactory for either half, although the detail was excellent. Two 7 inch×17 inch screens (Radelin T and UD), ratio 4:1, gave very satisfactory results (Fig. 1, 2 and 3). However, in women with above average sized fetus and uterus or in above average sized women, the 4:1 differential was not sufficient. Two 7 inch×17 inch screens (Radelin TF and UD), ratio 8:1, have proved to be excellent for this group (Fig. 4).

Since the anatomic density of the uterus cannot be divided sharply in half to coincide precisely with two 7 inch×17 inch screens, an attempt was made to fit the areas of differential speeds to the actual anatomy. Accordingly, a cassette was fitted with two screens (Patterson Hi and Detail), ratio 6:1, the former occupying the posterior one-third of the cassette, the latter occupying the anterior two-thirds (Fig. 5). Although very adequate and diagnostic roentgenograms were obtained, this was

TABLE I

Radelin	Exposure Factor	Relative Speed	Patterson
High Speed (TF)	0.5	8	
		6	Hi
General Purpose (T)	1.0	4	Par
High Resolution (HR)	2.0	2	
Ultra Detail (UD)	4.0	1	Detail

* From the Department of Radiology, Beth Israel Hospital, New York, New York. Presented as an Exhibit at the Fifty-ninth Annual Meeting of the American Roentgen Ray Society, Washington, D. C., September 29–October 3, 1958.

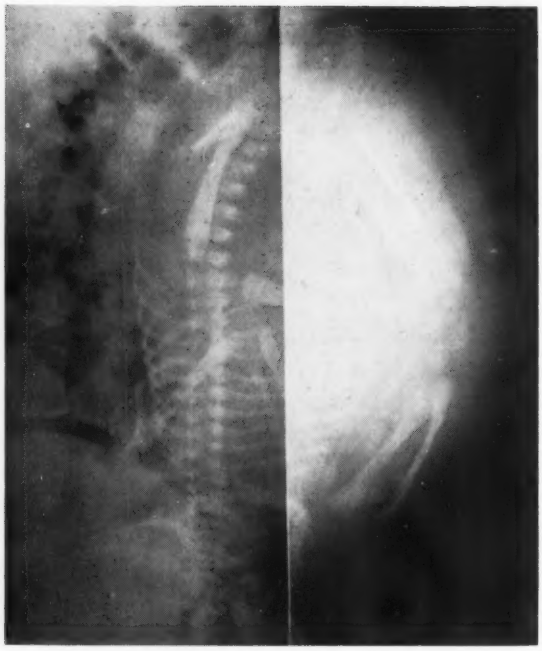

FIG. 1. Two screens, 7 inch×17 inch, ratio 4:1 (Radelin T and UD). Placenta on anterior wall. Excellent detail of fetus in both halves.

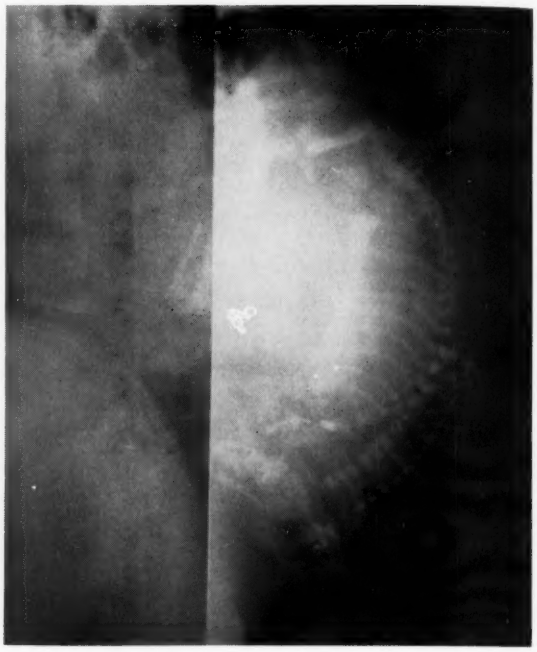

FIG. 3. Two screens, 7 inch×17 inch, ratio 4:1 (Radelin T and UD). No placenta on anterior or posterior wall. Fetal head displaced from sacrum. Proved placenta previa. Excellent detail of fetus.

FIG. 2. Two screens, 7 inch×17 inch, ratio 4:1 (Radelin T and UD). Placenta on posterior wall and fundus. Excellent detail of fetus (abnormal skull); femoral and tibial epiphyses not present.

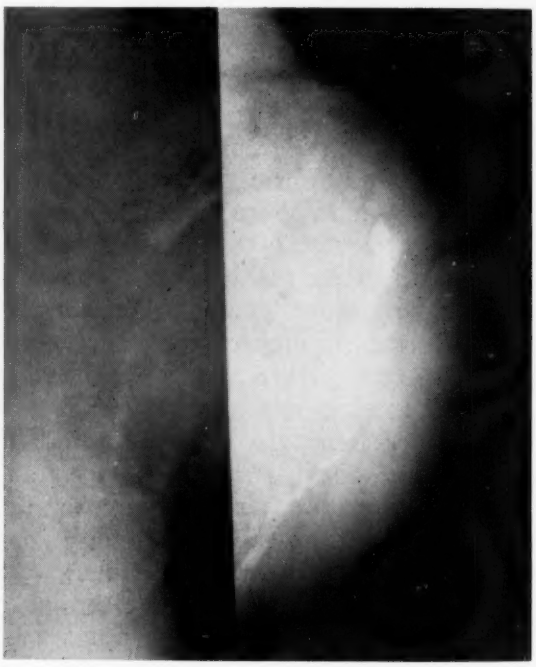

FIG. 4. Two screens, 7 inch×17 inch, ratio 8:1 (Radelin TF and UD). Placenta on anterior wall. Good exposure for both halves. The femoral epiphysis can be seen on the roentgenogram.

not completely satisfactory inasmuch as the variations in density of the fetus and soft tissues of the uterus could not be accurately reproduced in the anterior two-thirds.

The next step was to divide the cassette into thirds, in order to be able to give a more differential effect throughout all of the uterus. This was done with two cassettes. One cassette was made with three screens (Patterson Hi, Par, and Detail), ratios 6:4:1, each occupying only one-third of the total area of the cassette ($3\frac{1}{2}$ inch × 17 inch). The second cassette was made with a similar arrangement of three Radelin screens (TF, T, and UD), ratios 8:4:1. The roentgenograms obtained with these two combinations were excellent (Fig. 6, 7, 8 and 9). If only one cassette is available, the 8:4:1 ratio would probably be the more desirable, as it will prove better for the larger fetuses and larger mothers.

The sharp dividing lines of the margins of the different screens are a disadvantage. I have suggested to the manufacturers the desirability of an intensifying screen of

Fig. 5. Two screens, two-thirds and one-third of cassette, ratio 6:1 (Patterson Hi and Detail). Placenta on anterior wall. Excellent exposure for both anterior and posterior portions.

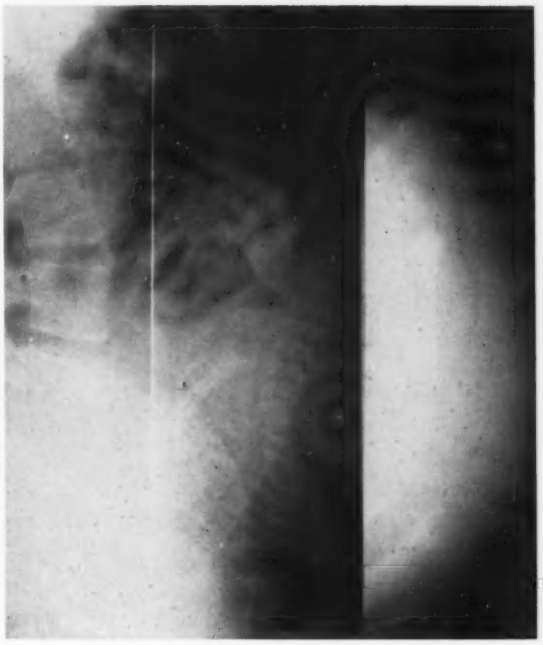

Fig. 6. Three screens, $3\frac{1}{2}$ inch × 17 inch, ratios 6:4:1 (Patterson Hi, Par, and Detail). Placenta on anterior wall. Excellent detail of soft tissues and fetus.

Fig. 7. Three screens, $3\frac{1}{2}$ inch × 17 inch, ratios 6:4:1 (Patterson Hi, Par, and Detail). Placenta on posterior wall and fundus. Excellent detail of soft tissues and fetus.

FIG. 8. Three screens, $3\frac{1}{2}$ inch×17 inch, ratios 8:4:1 (Radelin TF, T, and UD). Placenta low, anterior marginal previa. Head floating, displaced from pubis. Excellent detail of fetus and soft tissues (latter seen better on original roentgenogram).

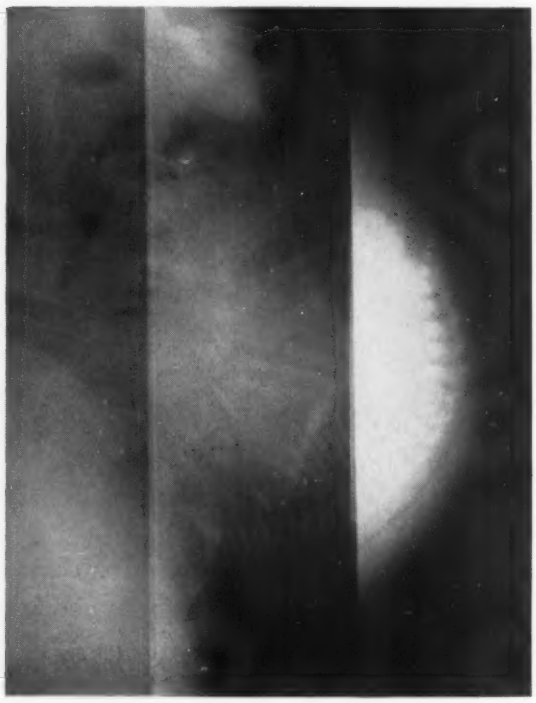

FIG. 9. Three screens, $3\frac{1}{2}$ inch×17 inch, ratios 8:4:1 (Radelin TF, T, and UD). Low anterior placenta. Excellent detail of fetus and soft tissues on original roentgenogram, partially lost on reproduction. Soft tissue density posteriorly due to fibroids. Placental site confirmed at cesarean section.

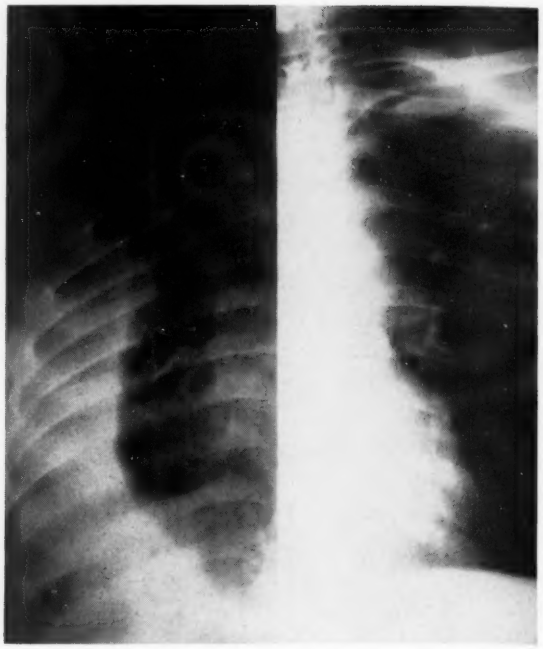

FIG. 10. Two screens, 7 inch×17 inch, ratio 8:1 (Radelin TF and UD). Anteroposterior Bucky roentgenogram of thorax. Despite dense thickened pleura on right, same exposure results in a single diagnostic roentgenogram for both halves of the chest.

differential speeds on the same base, giving a gradual change rather than a sharp demarcation. This appears to be impossible to accomplish with present technical knowledge.

This technique is applicable to other examinations. It can be used for chests with marked differences in density of the two hemithoraces (Fig. 10). A single lateral roentgenogram of the thoracic spine can also show lungs, ribs, and sternum (Fig. 11). A single anteroposterior roentgenogram of the chest is adequate for both the thoracic spine and for the ribs (Fig. 12). An anteroposterior lumbar spine and intravenous pyelogram can be obtained with the same exposure, provided the middle third of the

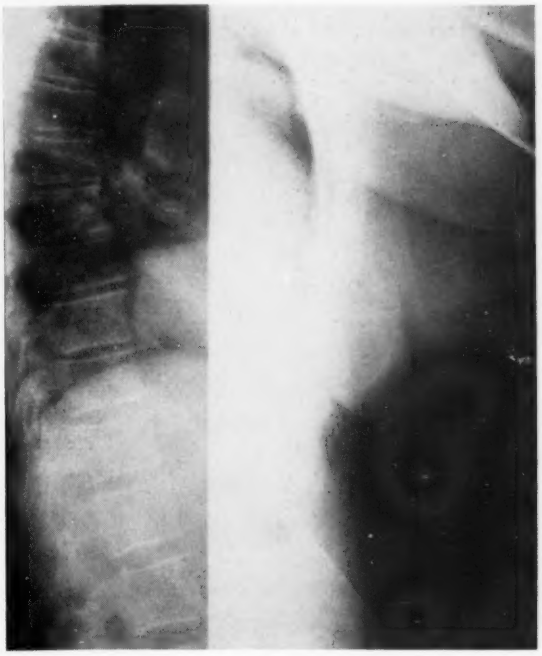

Fig. 11. Two screens, one-third and two-thirds of cassette, ratio 6:1 (Patterson Hi and Detail). Lateral Bucky roentgenogram of thoracic spine also diagnostic for lungs, ribs, and sternum.

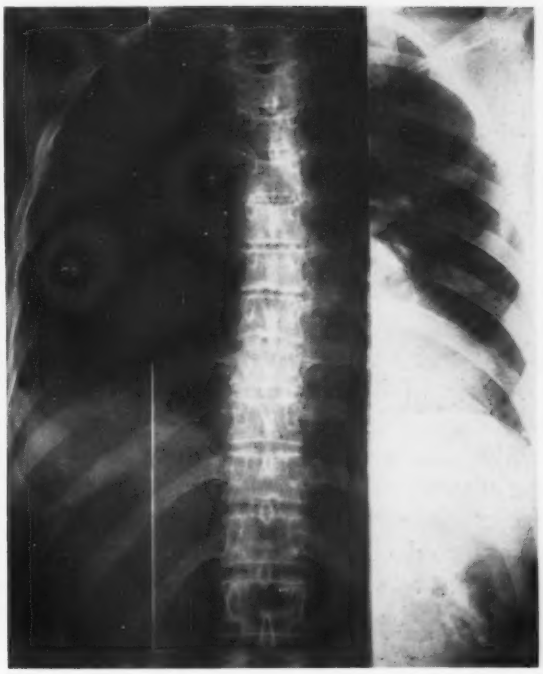

Fig. 12. Three screens, $3\frac{1}{2}$ inch×17 inch, ratios 6:4:1 (Patterson Hi, Par, and Detail). Anteroposterior roentgenogram of thoracic spine is also diagnostic for the left ribs.

cassette has the high speed screen with the slower speed screens on the lateral thirds. A single lateral roentgenogram of the abdomen may be adequate for both the lumbar spine and the soft tissues.

SUMMARY

1. Single film lateral placentography has been performed by the use of combinations of portions of intensifying screens of different speeds in one cassette.

2. The most advantageous combinations are indicated.

3. This technique is also applicable to other anatomic areas.

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The Radelin screens were generously supplied by the Radelin Division, United States Radium Corporation, Morristown, New Jersey. The Patterson screens were generously supplied by the E. I. DuPont de Nemours and Company. The representatives of both companies furnished able assistance in the preparation of the various combinations.

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AN INVESTIGATION OF EFFECTS OF OPAQUE MEDIA ON THE LUNGS WITH COMPARISON OF BARIUM SULFATE, LIPIODOL AND DIONOSIL*

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IN THE investigation of patients who have difficulty in swallowing, and particularly of infants with the symptom of choking during feeding, it is extremely likely that opaque medium injected into the esophagus or given by mouth will enter the tracheobronchial tree. It is important to try to decide which is the best opaque medium to use in the investigation of such patients. The swallowing mechanism must be examined and a fistula between the trachea and esophagus must be diagnosed or excluded. Such a fistula may be congenital or acquired. In the absence of a fistula, difficulty in swallowing with choking on feeding may be due to weak or paralyzed pharyngeal muscles or muscles of respiration, to impairment of consciousness, mental deficiency, or general weakness and debility. Esophageal obstruction with spill into the larynx from the overfilled esophagus, laryngeal paralysis or anesthesia, pharyngeal and laryngeal neoplasms, and very rarely congenital cleft larynx are other possible causes.

The choice of media lies between barium sulfate suspended in water, the aqueous suspension of dionosil, or an oily medium such as lipiodol or dionosil oily.

For this type of examination, barium sulfate has certain advantages. These include inexpensiveness, very high radiopacity, ease of handling, and the fact that barium sulfate is less likely than oily media to be aspirated during swallowing by normal patients.[29,66] Many radiologists still feel that barium sulfate is more dangerous than other opaque media when aspirated into the tracheobronchial tree. It is, in fact, noted in textbooks of radiology and chest diseases as being contraindicated under these conditions.[22,60,86,88]

In spite of much interest in the effect of opaque media on the tracheobronchial tree and lungs, and in spite of considerable experimental data accumulated in humans and in various animal species from work with these opaque media, we have been unable to find in the recorded literature any comparison of barium with lipiodol and dionosil in the same species under suitably controlled conditions. Our interest was particularly attracted to this problem by the following case.

REPORT OF A CASE

M.S. This female infant was born at full term after a normal pregnancy and delivery. The first feeding, which was glucose and water, was given at eight hours and was taken without difficulty, as were the next two feedings. On the second day of life, "excess mucus" was noted and there was evidence of bilateral pneumonia on physical examination. The abdomen was distended. On the third day of life, the infant appeared to be in extreme distress and her color was poor. The chest was reported to be clear on examination. On the fourth day of life, two feedings were attempted and on both occasions the infant became cyanosed. Esophageal atresia had been excluded by passage of a catheter into the stomach, so a provisional diagnosis of tracheo-esophageal fistula was made and the child was transferred to The Montreal Children's Hospital. On admission she appeared sick and lethargic. Shortly after

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Fig. 1. Authors' case. Tracheo-esophageal fistula (arrow) demonstrated at C6–C7 level following injection of opaque medium into the esophagus. Age four days.

admission, roentgenographic examination was undertaken to determine the presence or absence of tracheo-esophageal fistula. Lipiodol was injected into the esophagus by a nasal catheter. The opaque medium was seen to enter the trachea in small amounts, but the site of entry could not be determined with certainty under the fluoroscopic screen, or on the roentgenograms taken during the procedure. In view of the relatively frequent aspiration of injected or ingested lipiodol in normal infants without fistula, the injection was repeated with barium sulfate. The opaque medium again entered the trachea and it appeared likely that this was through a fistula situated just below the superior thoracic aperture, at the level of C6–C7 vertebrae. This was still felt to be uncertain and a third injection was undertaken, this time with lipiodol. A very small fistula was identified (Fig. 1). The opaque medium entered the right bronchial tree almost exclusively (Fig. 2).

The following day thoracotomy was done, the fistula identified, ligated and transected without difficulty. The postoperative course was satisfactory for the first eight to ten hours.

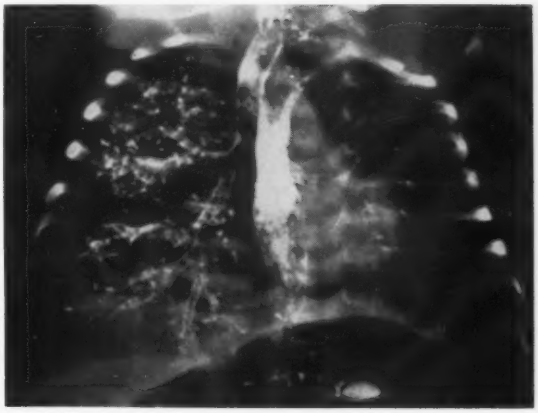

Fig. 2. Same case as in Figure 1. Opaque medium has entered the trachea and right bronchial tree, with slight alveolar filling. Age four days.

However, on the day after operation respiratory distress reappeared, with marked difficulty in clearing secretions from the upper respiratory tract. Two days postoperatively the child's condition progressively deteriorated, cardiac arrest occurred, and in spite of cardiac massage, death followed. Postoperative roentgenograms had shown partial clearing of the opaque medium which had entered the right lung, but a small amount of opaque medium was retained, and was scattered fairly uniformly throughout the right side, with little or none on the left side (Fig. 3).

On postmortem examination, the fistula was found to have been satisfactorily closed and transected. Both lungs were airless.

Histologic examination was reported as fol-

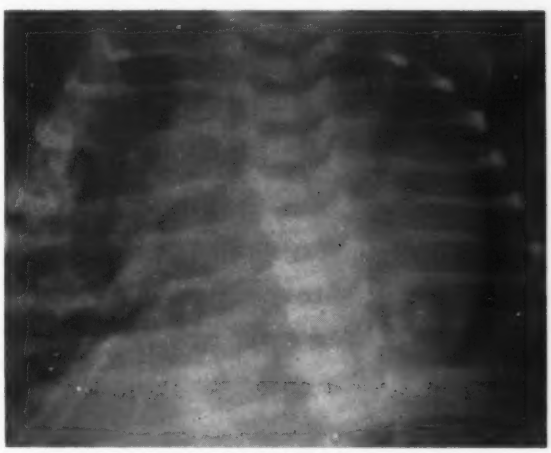

Fig. 3. Same case as in Figures 1 and 2. Small amount of retained opaque medium scattered through the right lung parenchyma. Age six days (death occurred later the same day).

lows (Dr. Ilona Kerner*): The right main bronchus showed signs of irritation in lining mucosa with metaplastic changes and inflammatory cell infiltrate. The left main bronchus showed one small focus of mucosal change. The right lung showed extensive bronchitis, bronchiolitis and alveolar pneumonia in all three lobes. Crystalline doubly refractile material in small to moderate amounts was scattered throughout the alveoli and alveolar ducts. One bronchus in the right upper lobe was plugged by the crystalline material. There were signs of mucosal irritation in two larger intrapulmonary bronchi. Marked congestion was present. Special stains (Felton method)[37] for lipiodol showed the lipiodol scattered throughout approximately the same areas as contained the crystalline material. The distribution of the two foreign substances corresponded fairly well in a general way. It was not possible to determine whether the inflammatory reaction was more pronounced about the crystalline material or the lipiodol, or in fact was caused by either.

The left lung showed absence of crystalline material and almost complete absence of lipiodol and absence of acute inflammatory cell reaction. Multiple small patchy areas of collapse were present. There were intra-alveolar and interstitial hemorrhages, and scattered collections of macrophages in the alveoli.

Comment. Although this infant's condition was poor at the time we first examined her on the fourth day of life, she appeared to withstand the operative procedure well, but died on the second postoperative day. The injection of opaque medium into the esophagus was followed by its aspiration into the trachea and into the right bronchial tree. Although the medium was coughed up out of the main and segmental bronchi, there was obvious peripheral retention in the right lung. The histologic sections showed both barium and lipiodol in the right lung and the reaction on the right side was more severe than that on the left. The comparison of the changes in the two lungs was made more difficult by the fact that the thoracotomy had been done on the right side.

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It is evidently quite impossible to attribute death to any one factor in such a child. Nevertheless, it is fitting to inquire whether the most innocuous opaque medium was used.

METHODS AND MATERIALS

Barium sulfate in tap water, dionosil aqueous, lipiodol, and tap water were injected into the tracheobronchial tree of rats under identical conditions. Roentgenograms were made following injection and immediately after sacrifice. The animals were sacrificed at intervals from six hours to four months and the roentgenographic and histologic findings compared.

We attempted injection of the contrast medium directly into the trachea through the larynx after the method of Huston *et al.*[51] but it was unsatisfactory in our hands. The animals tended to die with or without anesthesia during the necessary manipulation, apparently from suffocation. Percutaneous intratracheal needle puncture was attempted, but was not found satisfactory because of the very small size of the animal's trachea. Exposure of the trachea and injection of the medium directly into its lumen by needle puncture was also found to be unsatisfactory, apparently due to the small size of the tracheobronchial lumen, and suffocation of the animal occurred even with small amounts of the fluid medium. Similarly, tracheotomy and direct injection by polyethylene catheter into the trachea was not successful.

Several experiments were done with nebulized lipiodol and barium sulfate in water suspension, using various sizes of droplets in the mist or spray produced, with the animals in open and enclosed boxes, and direct spraying of the mouth and pharynx with the nebulized medium was also attempted. These direct and indirect spray and mist methods were uniformly unsuccessful. The degree of opacity of the tracheobronchial tree, as shown by fluoroscopy and roentgenograms, was so slight as to be of no value in this type of investigation. This was presumably due to

very active and efficient upper respiratory filtering and ciliary action. The opaque medium was swallowed and could be identified in the intestinal tract but was not retained in the respiratory tract in demonstrable amounts.

The method found satisfactory, and which was employed throughout the remainder of the experiment, was as follows: Norwegian hooded rats, Royal Victoria strain, weighing 150 to 200 grams and approximately two to three months of age were used. The animals were anesthetized with intraperitoneal injections of nembutal sodium, 50 milligrams per cc., in dosage of 4 milligrams per 100 grams of body weight. Following the induction of general anesthesia by this injection, the trachea was exposed in the neck as rapidly as possible, a small hole made on its anterior surface, and a fine polyethylene catheter passed down the trachea and blindly into whichever main bronchus it entered until it was arrested (Fig. 4 and 5). At this level, 0.05 cc. of the opaque medium was injected, the catheter was withdrawn, and the incision in the neck closed. The animals were then roentgenographed as soon as possible, preferably before recovery from the anesthesia. Sacrifice was by a lethal dose of 1 cc. of nembutal injected intraperitoneally. The sacrifices were made at intervals of six hours, one day, one week, one month and four months after the intrabronchial injection. Immediately upon death and before postmortem examination, 66 of the rats were again roentgenographed. This included all

Fig. 4. Method of injecting opaque medium into anesthetized rat's tracheobronchial tree.

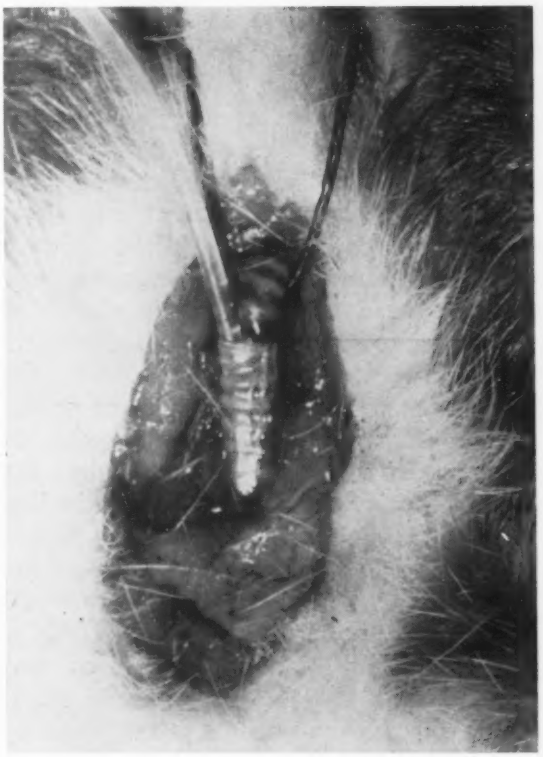

Fig. 5. Close-up of trachea with polyethylene catheter entering small hole cut in anterior wall of trachea.

animals sacrificed at four months. In each of the four groups (barium sulfate, lipiodol, aqueous dionosil, and tap water), a minimum of 5 animals were used for each of the sacrifice periods. In addition, 9 animals were left in cages, without having had anesthesia or operation of any kind, to serve as controls. These controls were sacrificed at twenty-four hours, seven days, one month and four months.

As soon as possible after the death of the animal, the trachea was exposed in the neck, ligated, and sectioned above the ligature. The thorax was then opened and the heart, lungs and the tracheobronchial tree removed as one specimen and immediately fixed in a solution of cobalt formalin for a minimum of forty-eight hours. Sections were then made through each lung, attempting in each case to transect the injected area in one lung and a similar area in the other lung to serve as a control. The area from which the section

was taken was determined partly by the roentgenogram made immediately after the injection and partly by careful examination of the gross specimen itself.

Microscopic sections were prepared by staining with hemalum-phloxine-saffron. In addition, sections were made from the lung specimens of 2 lipiodol injected animals killed at six hours and 2 at one hundred and twenty days, and from 2 animals killed at six hours after water injection. These six sections were stained differentially for lipiodol according to the method described by Felton.[36] All microscopic sections were examined by one of us (J.S.) and graded 1 to 4 in terms of edema, polymorphonuclear response, mononuclear response, necrosis, and fibrosis. For this purpose lymphocytes, plasma cells and macrophages were grouped together as mononuclear cells. Grade 1 represented minimal change, which was often normal. Grade 2 represented slight change which was borderline or generally abnormal. Grade 3 was a moderate to marked change, always abnormal, and Grade 4 represented severe abnormality. In addition, wherever foreign material was identified, it was also graded as 1 plus to 4 plus.

Near the end of the experiment it was found that, in error, the aqueous suspension of barium had contained sucrose in approximately 10 per cent solution. This was the preparation of barium used routinely to give our infant patients. It was therefore the preparation used in the case reported above. It is prepared under sterile conditions in the infant formula room. This concentration of sucrose produces a solution which is approximately isotonic. It appeared essential, however, to determine whether it would aggravate or ameliorate any possible effect of the injected barium. A fifth group of animals was therefore injected, using barium sulfate in tap water. The barium was approximately 50 per cent weight by volume in this preparation, as well as in the sucrose solution.

In the complete study, a total of 187 rats were used. The animals were kept until the time of sacrifice in cages under identical conditions, identified by ear tags. Animals of the four different groups were not segregated according to the opaque medium used.

TABLE I

DEATHS DURING INJECTION (25 RATS)

	Died	Injected	Per Cent
Water	2	33	6
Barium	8	65	12
Lipiodol	7	43	16
Dionosil	4	33	12
Anesthetic only	4	—	—
Total	25	174	

RESULTS

Of the 174 rats injected, 25 died during the operative procedure. In 4 instances, the deaths occurred before the opaque medium was injected. In some of the animals, in spite of the bronchial rather than tracheal site of the injection, there was a tendency to suffocation during the procedure. A further group of 13 died of pulmonary or mediastinal infection at intervals varying up to thirty-five days. The analysis of the deaths, while included in this presentation (Table I and II), is not considered as highly significant. A progressive improvement in technique is suggested by the fact that these deaths from infection occurred in the animals used in the first half of the experiment only. Massive changes were observed at postmortem examination in 6 animals sacrificed, including lung

TABLE II

DEATHS FROM INFECTION (13 RATS)

	Died	Injected	Per Cent
Water	2	33	6
Barium	4	65	6
Lipiodol	7	43	16
Dionosil	0	33	0
Total	13	174	

abscess and pulmonary cyst. These findings in a small group were not considered as significant in terms of the opaque medium itself, but rather as complications of the tracheotomy and possibly of the intraperitoneal anesthetic injection, and therefore similar to the 13 animals which died of infection. One of the 9 control rats which had no procedure done showed an extensive lung abscess at the time of sacrifice.

ROENTGENOGRAPHIC FINDINGS

Lipiodol (Fig. 6). At six hours the injected segment tended to show massive consolidation and sometimes collapse. The opaque medium present diminished progressively to one month. None of the opaque medium remained at the end of four months except in one animal, where it was present in a moderate amount.

Barium (Fig. 7). The consolidation of the lung by the opaque medium tended to be a little greater six hours after the injection than with the other media, with little change at the twenty-four hour, seven day and twenty-eight day examination. At four months opaque medium had not completely disappeared from the lung.

Dionosil (Fig. 8). The opaque medium had diminished slightly in six hours, was approximately half gone in twenty-four hours, and in seven days had disappeared in all cases. No dionosil could be identified roentgenographically at one month or four months.

Water (Fig. 9). No roentgenologic abnormality was demonstrated except in the animals which died and in one animal sacrificed at four months, which showed an upper lobe pneumonia.

Summary of Roentgenographic Findings: Water produced no consistent change, dionosil was uniformly reabsorbed within seven days and reabsorption had begun at six hours. Lipiodol was diminished in amount but still present at one month, and had disappeared in four months. Barium had diminished only slightly in one month and had not entirely disappeared in four months, using a suspension of barium in tap water.

MICROSCOPIC FINDINGS

Controls. The control animals showed polymorphonuclear and mononuclear cells in sufficient numbers to be graded as 1 to 2 in the majority of the animals. No edema was observed and minimal fibrosis (Grade 1) was seen in only one of the 9 control animals examined. There was no necrosis.

Water. The cellular response, both polymorphonuclear and mononuclear, was slightly greater than that observed in the control animals. It reached its maximum at

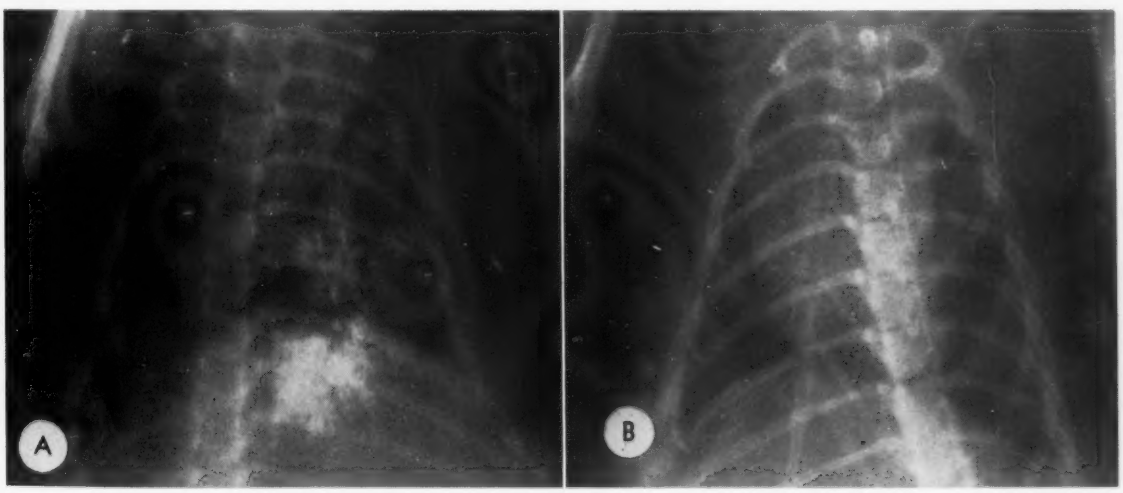

Fig. 6. (*A*) Roentgenogram taken immediately after lipiodol injection. (*B*) Roentgenogram taken four months after lipiodol injection. No opaque medium remains.

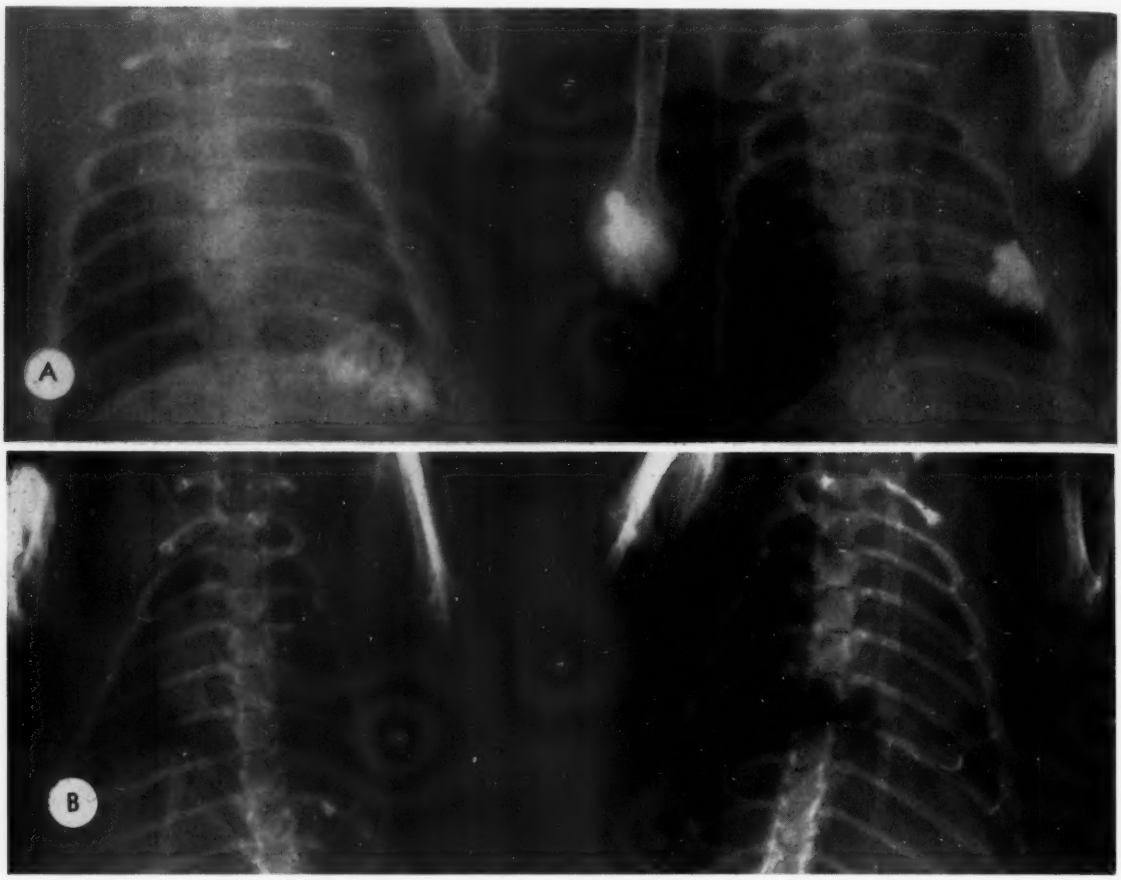

FIG. 7. (*A*) Roentgenograms taken immediately after barium sulfate (in tap water) injection. (*B*) Roentgenograms taken four months after injection of same suspension. A small amount of opaque medium remains.

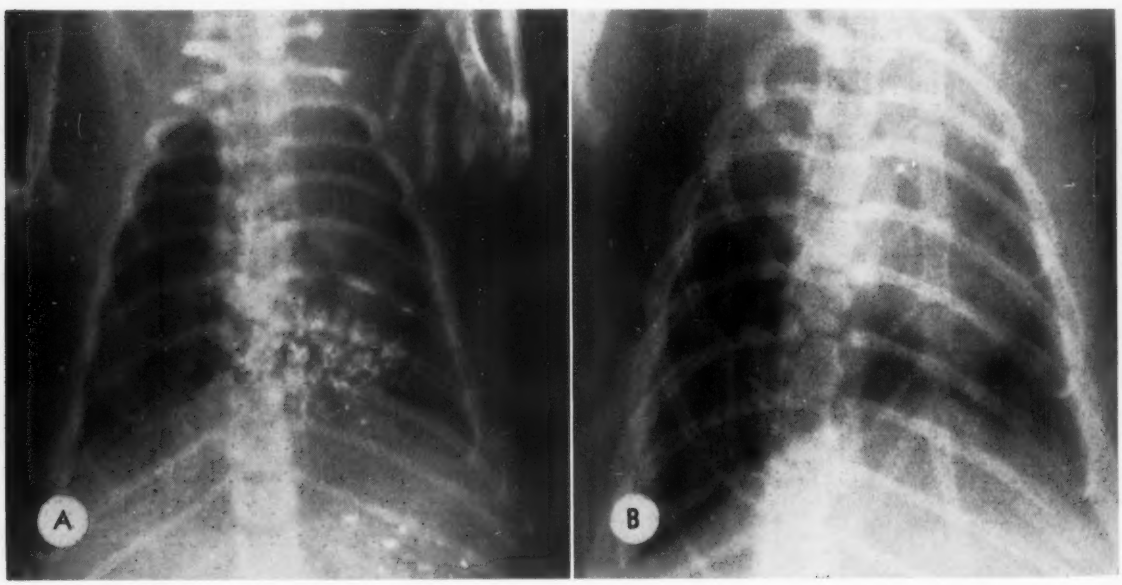

FIG. 8. (*A*) Roentgenogram taken immediately after dionosil injection. (*B*) Roentgenogram taken four months after dionosil injection. No opaque medium remains.

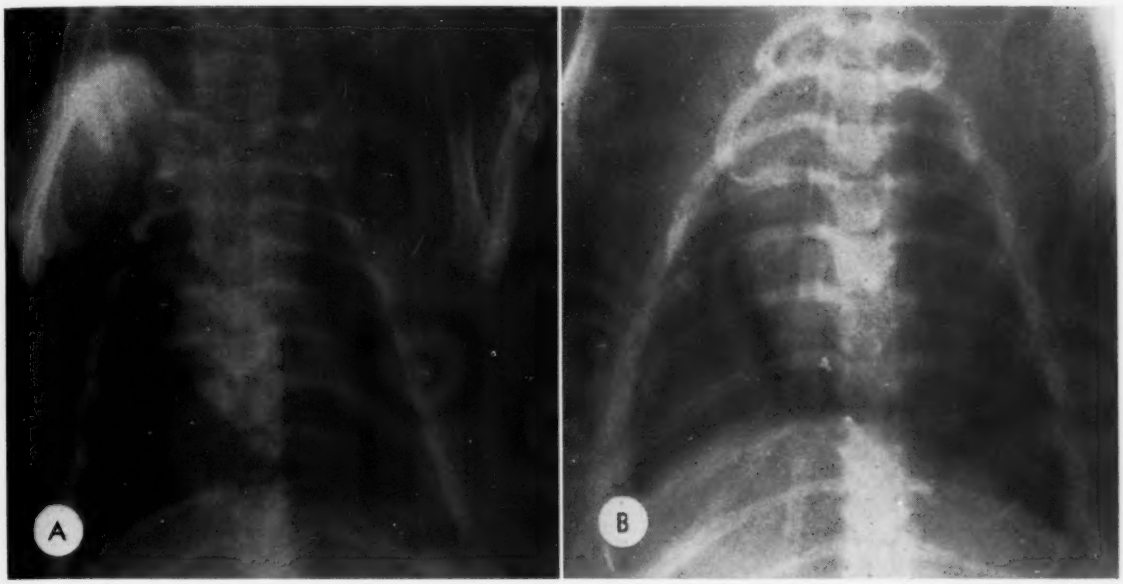

Fig. 9. (*A*) Roentgenogram taken immediately after water injection. (*B*) Roentgenogram taken four months after water injection.

twenty-four hours and seven days and continued at approximately the same level throughout the remainder of the experiment, up to four months. It was generally slight (Grade 2). Edema and necrosis were not observed and fibrosis was present in minimal degree in only occasional animals at each of the sacrifice periods.

Barium plus Sucrose (Fig. 13, *A*, *B* and *C*). Moderate to marked edema was present at six hours and twenty-four hours, and subsiding almost completely by seven days, never to reappear. Polymorphonuclear response was Grade 2 to Grade 3 at six hours, increasing to Grade 3 to Grade 4 at twenty-

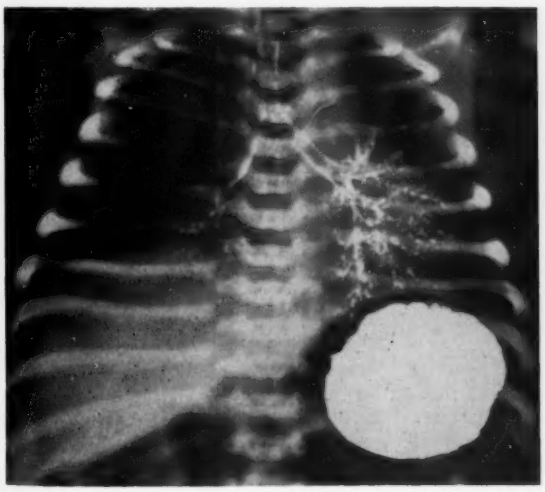

Fig. 10. Infant with no evidence of tracheo-esophageal communication. Lipiodol given by mouth for investigation of vomiting was aspirated into tracheobronchial tree. This is not an infrequent occurrence with oily opaque media.

Fig. 11. Lipiodol injected into nares to exclude choanal atresia (not found). Lipiodol readily entered trachea and main bronchi.

FIG. 12. Barium paste given by mouth during heart fluoroscopy. A small amount was aspirated into the trachea. This is an unusual occurrence.

four hours, decreasing at seven days and twenty-eight days, and virtually resolved by four months. There was a similar mononuclear response. Necrosis was not present up to one month and was present in minimal degree in 2 out of 6 animals at four months. Fibrosis was present minimally in only a few animals.

Lipiodol (Fig. 14, *A*, *B* and *C*). Edema was absent at six hours, mild (Grade 2 to Grade 3 in one-half of the animals) at twenty-four hours, and thereafter absent. Cellular reaction was minimal at six hours, and moderate at twenty-four hours and seven days, becoming slight at twenty-eight days and four months. Necrosis was absent in the first week, appeared in a minimal degree at one month, and had virtually disappeared by four months. Fibrosis appeared in a minimal degree at twenty-eight days and four months.

Dionosil (Fig. 15, *A*, *B* and *C*). Edema appeared at six hours in moderate degree. It diminished slightly by twenty-four hours and was essentially gone by seven days and did not reappear. Similarly, a moderate inflammatory cellular response appeared at six hours, remained unchanged at twenty-four hours and seven days, diminished at twenty-eight days, and decreased to minimal levels at four months.

Barium. Edema was present in mild degree in only 1 animal at six hours and was not observed at any other time. Cellular reaction was slight at six hours, increased to moderate at twenty-four hours, and was moderate, then slight, throughout the remainder of the observation period.

Summary of Microscopic Findings: Dionosil aqueous produced moderate and definite edema at six hours, which gradually subsided. Cellular reaction was moderate for the first twenty-four hours.

Lipiodol produced only slight initial change but the cellular reaction, though mild, was at least as great as with the other injected media, and the necrosis and fibrosis at four months, though still slight, were greater than with the other media.

Barium in tap water produced a moderate inflammatory change during the early stages, and no late necrosis or fibrosis. There was no significant edema at any time.

Barium in sucrose solution produced inflammatory cellular response in the later stages, and, in particular, early edema.

The barium sulfate was identifiable as crystalline doubly refractile material in most of the animals injected with barium sulfate in pure tap water. Macrophages in the vicinity of barium deposits generally showed brownish granular staining of the cytoplasm.

The special staining done for lipiodol in 2 of the animals killed early (six hours) and 2 of the animals killed late (four months) showed the lipiodol to be present in moderate amounts early, minimal or absent later, corresponding, in this small sample of the group, to its disappearance

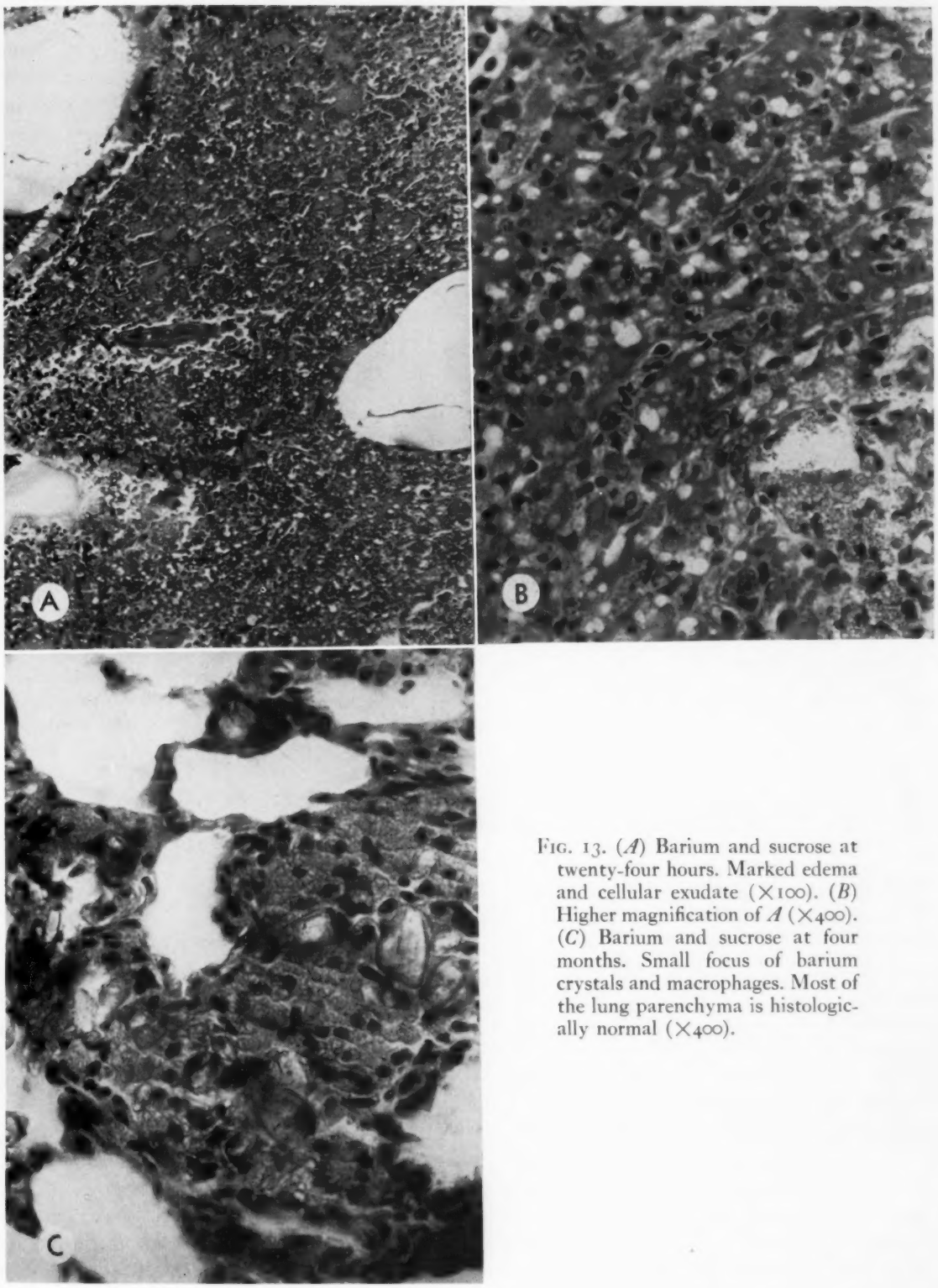

Fig. 13. (*A*) Barium and sucrose at twenty-four hours. Marked edema and cellular exudate (×100). (*B*) Higher magnification of *A* (×400). (*C*) Barium and sucrose at four months. Small focus of barium crystals and macrophages. Most of the lung parenchyma is histologically normal (×400).

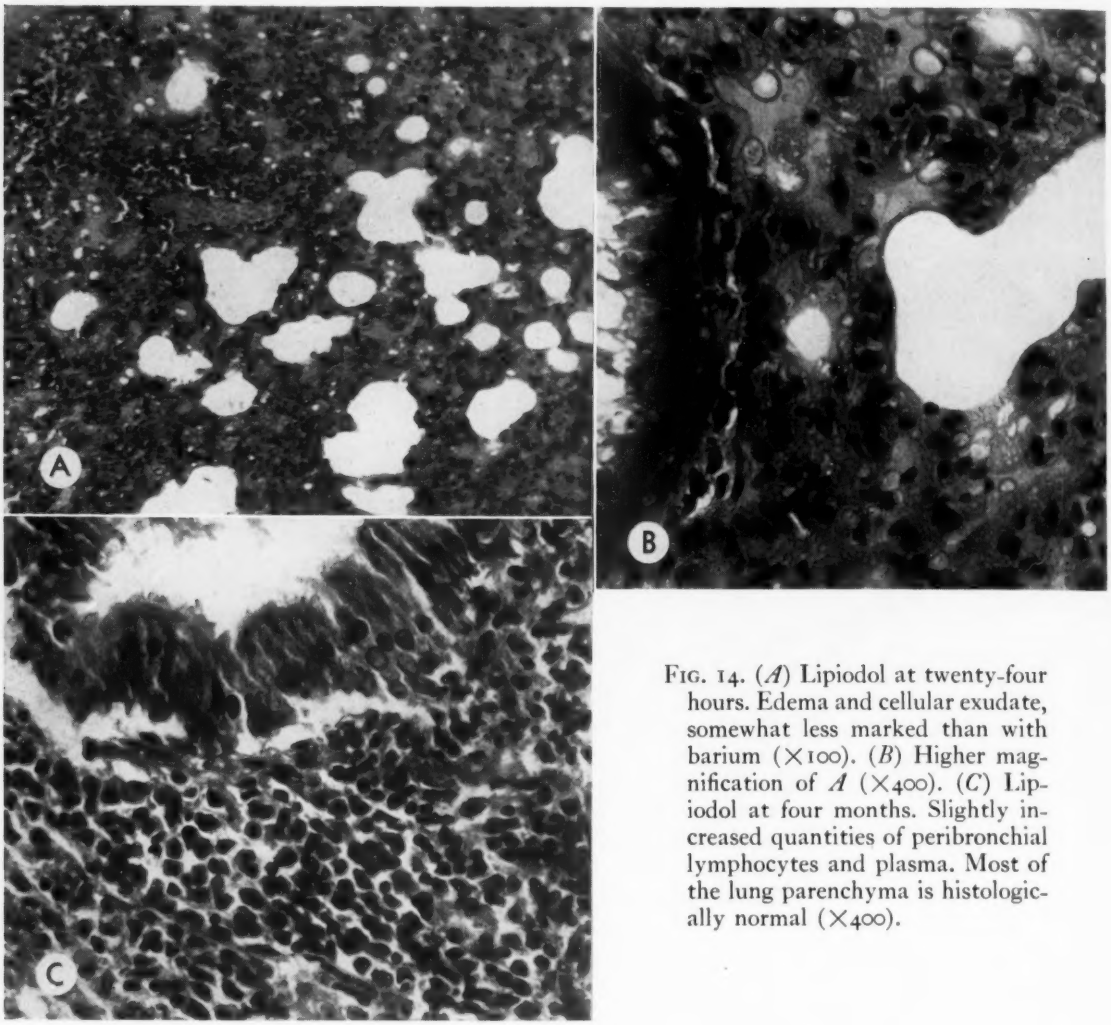

FIG. 14. (*A*) Lipiodol at twenty-four hours. Edema and cellular exudate, somewhat less marked than with barium (×100). (*B*) Higher magnification of *A* (×400). (*C*) Lipiodol at four months. Slightly increased quantities of peribronchial lymphocytes and plasma. Most of the lung parenchyma is histologically normal (×400).

from the roentgenograms at four months. The validity of this observation and of Felton's observations, appeared to be confirmed by the absence of any similar staining material from the lungs of the animals injected with water. In the routine sections, the lipiodol could not be identified, since it would dissolve out of the preparation on the slides, but could sometimes be suspected by the presence of vacuoles.

The dionosil, or its components, could often be suspected by the presence of a gray or greenish-gray precipitate, which did not appear with any of the other media. However, since no special stain was used for propyliodone or CMC, this finding could not be confirmed.

REVIEW OF LITERATURE

The search for a safe radiopaque material to introduce into the respiratory passages for visualization of the tracheobronchial tree began in 1906, when Springer[94] attempted to carry out bronchography on dogs. Waters *et al.*[102] in 1917, again in animals, attempted bronchography using 10 per cent suspensions of iodoform in olive oil. They also attempted injections with thorium and bismuth. Chevalier Jackson,[53] in 1918, reported the first successful human bronchography. He insufflated dry bismuth subcarbonate through a bronchoscope to visualize the position of a foreign body in relation to the bronchial tree, using stereoscopic films. It is interest-

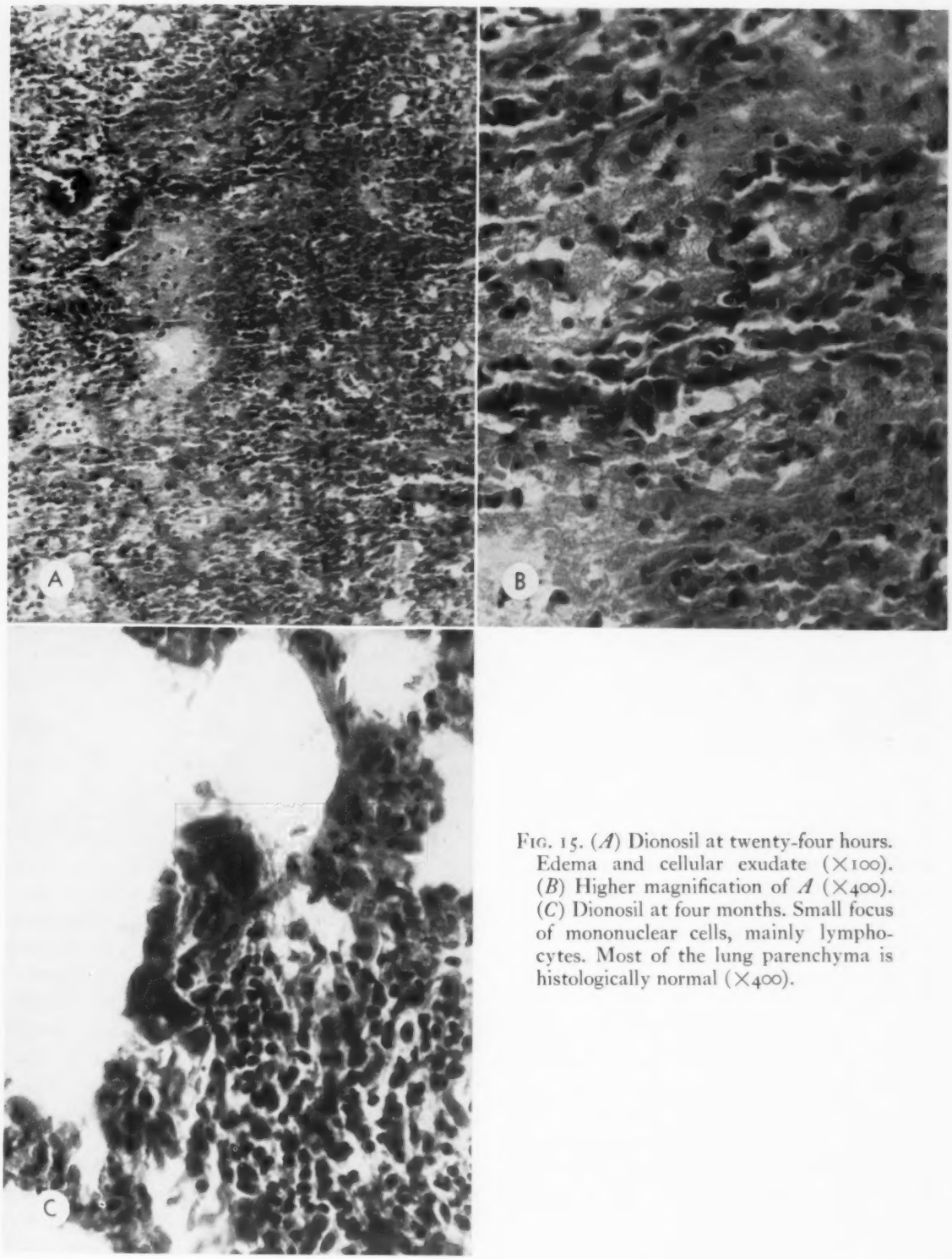

Fig. 15. (*A*) Dionosil at twenty-four hours. Edema and cellular exudate (×100). (*B*) Higher magnification of *A* (×400). (*C*) Dionosil at four months. Small focus of mononuclear cells, mainly lymphocytes. Most of the lung parenchyma is histologically normal (×400).

ing that as late as 1950 Jackson and Jackson[54] were still recommending bismuth in bronchography (both the subcarbonate and the subnitrate are mentioned, without any discussion as to which is superior), although they were placing more emphasis

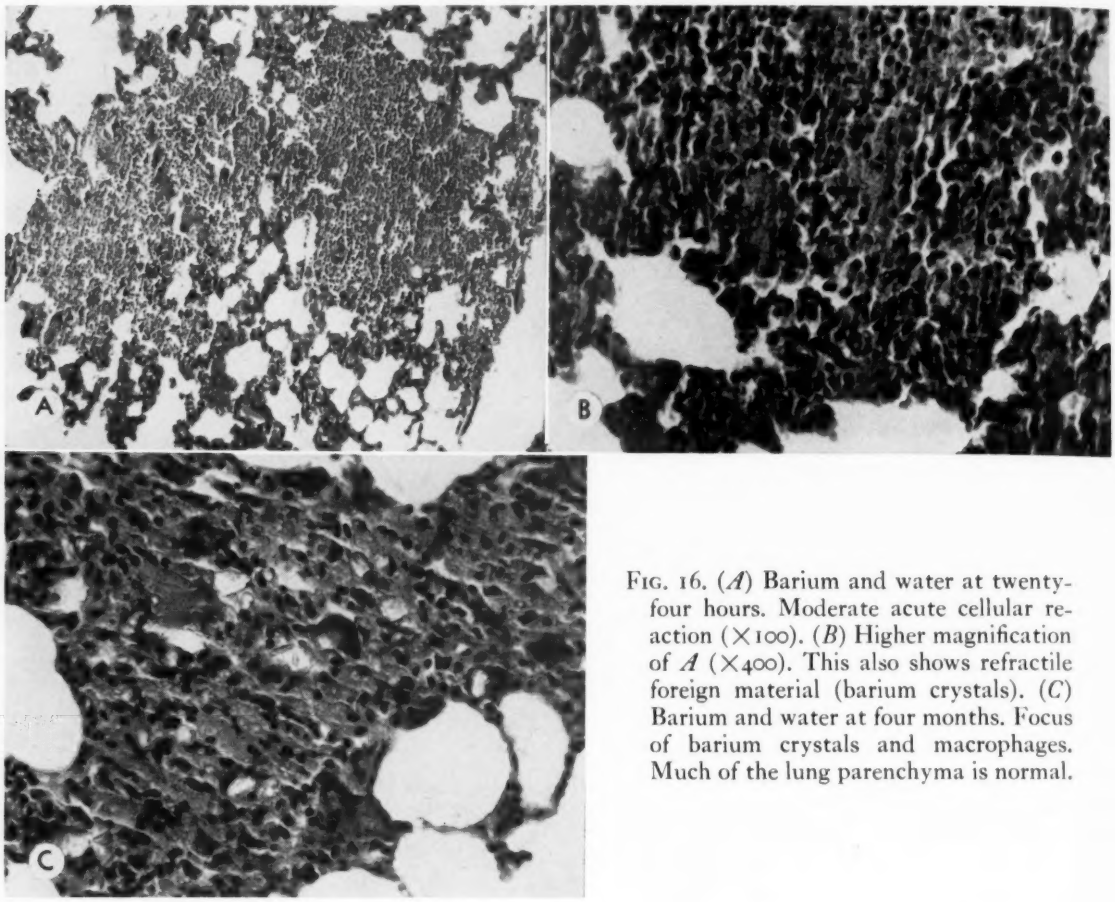

FIG. 16. (*A*) Barium and water at twenty-four hours. Moderate acute cellular reaction (×100). (*B*) Higher magnification of *A* (×400). This also shows refractile foreign material (barium crystals). (*C*) Barium and water at four months. Focus of barium crystals and macrophages. Much of the lung parenchyma is normal.

on lipiodol in routine procedures. The first mention of barium is by Bullowa and Gottlieb[21] in 1920. After trying barium suspended in gelatin and thorium, both without success, they used barium sulfate in olive oil mixed with smooth paste for bronchography in dogs, and reported that this mixture appeared to be relatively innocuous in healthy dogs. The plates showed rather massive filling of the bronchial tree, but it was well outlined. Only one dog in their group died. They also used barium sulfate in liquid petrolatum on one occasion and with success. They used both fluoroscopy and x-ray plates to study bronchial anatomy and physiology, but did not report any pathologic examination of the animals used. In 1921, Lynah and Stewart[61] injected a sterile (boiled) mixture of bismuth subcarbonate and pure olive oil to outline and localize lung abscesses and bronchiectasis in humans. They reported that this mixture seemed to be beneficial to the patients, and did no harm. Sicard and Forestier[92] in 1922 reported on the use of lipiodol in bronchography. It is noteworthy that they also recommended it for epidural and subarachnoid injections, in diagnosis and in some cases for treatment of central nervous system inflammatory disease. Similarly, in the bronchial tree, they were impressed by both the therapeutic and diagnostic possibilities of lipiodol injections. They were not the first, however, to consider the possibility of using iodized poppy seed oil for therapeutic intratracheal injections. Winternitz[108] in 1898 had reported on the use of this combination for endotracheal therapy. After Sicard and Forestier's paper in 1922, a tremendous field in chest diagnosis was opened up, and the high radiopacity and very low

toxicity of iodized oil were widely appreciated and exploited. It soon, however, began to be recognized that lipiodol bronchography was not entirely without hazard. From 1927 until the present time, a series of clinical and experimental investigations were published, reporting that while the opaque medium was without apparent harm in the vast majority of cases, it could cause anaphylaxis, acute iodism, lipid pneumonia, or lipid granuloma formation.[3,8,16,17,25,37,40,41,43,62,69,78,85,87,95,97,98,104,106,107,110] In addition, it had the considerable disadvantage of remaining in the lung for varying periods of time from a few hours to many months after bronchography, and therefore causing difficulty in the interpretation of subsequent chest roentgenograms.

Pinkerton,[75,76] investigating lipid pneumonia in infants and children in 1927 and experimenting on oils and fat in the lung with rabbits and puppies in 1928, concluded that the degree of pulmonary damage and fibrosis depends on the amount of free fatty acid originally present, and on the rapidity of hydrolysis of the fat in the lung. Brown[17] in 1928 injected lipiodol into cats' tracheas and showed that it excites a mononuclear infiltration, and that the large phagocytes are chiefly responsible for the removal of the lipid. The first report in the English literature of acute iodism following lipiodol bronchography was that of Scadding[85] in 1934; of 2 cases, one died with pulmonary edema and generalized sensitivity reaction, and one recovered after four weeks. This and subsequent reports showed that lack of sensitivity to previous potassium iodide therapy and lack of response to previous iodine sensitivity tests were no guarantee against this catastrophe. The problem of whether or not lipiodol can be safely administered in the presence of pulmonary disease, particularly tuberculosis, has given rise to considerable disagreement. In 1937, Schneider and Segal[87] reported a case of pulmonary tuberculosis developing at the site of retained lipiodol one year after the bronchogram. That this was anything more than coincidence was not established, and since then lipiodol has repeatedly been used in the presence of tuberculosis, without conclusive evidence of specific damage.[2]

In 1938 Titus *et al.*[99] experimented with opaque media for use particularly in uterosalpingography, and recommended a mixture of skiodan 40 per cent and acacia 20 per cent. They suggested that this would also be useful in bronchography, but others found that the acacia was too irritating, and the mixture never found wide acceptance.

Paterson[70] in 1938 investigated a variety of lipids of vegetable, mineral and animal origin as well as fatty acid esters, sterols, and phosphatides. His conclusions were similar to those of Pinkerton, and he emphasized the slow removal of oil droplets by phagocytic action. Granulomata and fibrosis were found least often after vegetable oils were used, greatest when animal oils were used and intermediate when mineral oils were used. Fractions of the oils, particularly the unsaturated oils, produced similar results.

It appeared that a water soluble opaque medium would be more suitable for bronchography if one could be found which was nonirritating, and in which the desired viscosity could be attained. The viscosity would have to be sufficient to prevent alveolar flooding by the medium during the bronchographic examination and to delay its absorption long enough to allow adequate fluoroscopic examination and roentgenography. To achieve the desired viscosity, pectin, dextrin and dextrans, alginate, polyvinyl alcohol and gum arabic had all been tried as thickening agents,[63,65,71,86] but had been found to be either too irritating to the bronchial tree, as in the case of polyvinyl alcohol, or to require a high concentration of the thickening agent. Morales and Heiwinkel[64,65] in 1948 introduced carboxymethylcellulose (CMC) and showed that in many respects it was the ideal material for this purpose. It is a cellulose ether, also known as cellulose

glycol acid ether and cellulose glycolate. Used as the sodium salt, it is easily soluble in water, and in concentrations as low as 1 to 3 per cent forms highly viscous solutions which are completely clear and colorless, do not gelatinize even on storing, and are nonirritating. CMC had previously been extensively investigated in animals of several different species and in humans,[49] and had been found to be completely nontoxic when given by mouth or when applied to skin, genitals or mucous membranes. In animals, experiments had been done with intravenous injections, and even by this route the medium had been shown to be surprisingly innocuous.[90] Morales and Heiwinkel[65] combined it with iodopyracet (diodone, which is marketed under various trade names including diodrast and umbradil) and, as umbradil-viskos B, containing 50 per cent umbradil, 2.7 per cent CMC, and 0.5 per cent xylocain, recommended it as a suitable opaque medium for bronchography.

Using slightly different percentages of CMC, they also produced umbradil-viskos H and umbradil-viskos U for hysterosalpingography and urethrography, respectively. They did extensive experiments on animals with intra-articular, peritoneal, pleural, subpericardial, subcutaneous and intravenous injections of this medium. In humans, intravenous, subcutaneous and intramuscular injections were done as well as urethrography and hysterosalpingography. They showed that, in addition to being of suitable radiopacity and viscosity, the mixture has a low surface tension so that it reaches small pockets and channels and spreads rapidly on large mucosal surfaces.

Similar preparations containing iodopyracet and CMC, but with slightly different proportions and viscosity ratings, were advocated by others, including Fischer.[86] Extensive trials of these water soluble contrast media with CMC followed.[6,13,20,45,46,71,72,86,112] In general, they were found to give satisfactory bronchial opacity but to be irritating to the bronchial mucosa in rather marked degree, necessitating very complete mucosal anesthesia for successful bronchography. This bronchial irritation appeared to be due largely to the hypertonicity of the aqueous solution.

In 1953, a radiopaque medium of low solubility in water was introduced by Tomich, Basil and Davis.[100] This was dionosil, the N-propyl ester of iodopyracet, in a 50 per cent W/V suspension in aqueous solution of CMC or in arachis oil, and called dionosil aqueous (30 per cent iodine) and dionosil oily (34 per cent iodine), respectively. Of these, the aqueous suspension, though isotonic, is more irritating and is cleared more rapidly from the lungs; the oily suspension is no more irritating than lipiodol, is cleared a little less rapidly but still within three to four days, and is a little more likely to cause alveolar filling. The radiopaque ester, propyliodone, common to both the oily and aqueous preparations, is hydrolized enzymatically in the lungs, absorbed, and is excreted as the sodium salt of diodone by the kidney. There is no breakdown to free iodine or inorganic iodides.

The fate of the CMC contained in dionosil aqueous (and other water miscible bronchographic media) is less clear. Tomich *et al.*[100] stated that it could not be demonstrated in the lungs of their rabbits after five days. Vischer,[101] using rats and guinea pigs, reported that CMC was retained in the lungs up to four months, and acted as a foreign body with production of giant cells, inflammatory cells, and fibrous tissues. He found that similar changes were demonstrable in human lungs up to ninety days after bronchography. Zollinger[112] disagreed with these conclusions, reporting that the material Vischer observed in the lungs was not CMC but mucin, and that the interstitial granulomas observed by Vischer were mucin granulomas, and could be found whether or not CMC had been used in animals, or in human specimens. Werthemann and Vischer,[105] using both the Feyrter and Hotchkiss staining techniques on animal lungs, insisted that granuloma-

tous and cellular reactions demonstrably occurred in response to retained CMC and that this was clearly different from the mucin reported by Zollinger. Björk and Lodin[13] carried out further experiments with umbradil-viskos B and dionosil aqueous as well as with dextran fractions of different molecular weights. They reported that CMC can cause granulomata in the lungs, though they showed this response to umbradil-viskos B, and not to dionosil. They raised the possibility that the formation of granulomata does not depend on chemical properties of the colloid, but rather on the physical properties of the large molecules of the carbohydrates.

The arachis oil used as a suspending agent in dionosil oily does not appear to be substantially different from poppy seed oil with respect to its effect on the lungs or its rate of disappearance from the lungs, as shown by Björk and Lodin[14] in 1957 and by the earlier workers experimenting with the various vegetable, animal and mineral oils. In dionosil oily, however, the iodine is not combined with the lipid, and the retention of the oil, if any, is not identifiable roentgenologically, as in the case of retained lipiodol.

Meanwhile, in 1945, the addition of a sulfonamide to lipiodol was recommended by Dormer *et al.*, in order to increase the viscosity of the iodized oil and thereby prevent the undesirable alveolar filling frequently seen with lipiodol. This mixture marketed as visciodol, has found increasing acceptance, particularly in Europe. Several enthusiastic reports have appeared, stressing the minimal alveolar filling, rapid elimination from the lungs, high radiopacity, and lack of irritation.[2,73]

In 1956, Shapiro[89] suggested cesium chloride for bronchography. He tried it in 20 dogs and found that in 30 per cent strength injections by tracheal catheter it passed rapidly into the alveoli and was absorbed completely rather quickly. CMC was then added and gave good results. He used it in 2 human cases, but it was followed by severe chest pain in the area examined. Resection the following day showed edema of the injected lobe.

Compared with the considerable experience with, and work on, the water soluble and the oily opaque media for bronchography, the effect of barium sulfate on the tracheobronchial tree and lungs has received much less attention. Following the successful experiments of Bullowa and Gottlieb[21] on dogs with barium in olive oil in 1920, the next report of experience in humans was that of Fishel[38] in 1923 in a case in which barium entered the lungs accidentally and apparently caused no trouble. In 1933, Arrigoni[5] injected a suspension of three different grades of material from barium mines into the tracheas of rats and rabbits. His intention was to try to assess the effect of the barite in different degrees of purity as recovered from the mines, compared with pure barium sulfate. It was known that the workers in the barite mines suffered from pneumoconiosis, but it was also known that in these cases silicosis played a part, and the contribution made by the barium as compared to the silica was not known. Arrigoni found evidence of bronchiolitis in the injected animals. Around the bronchioli and small arteries there were numerous round formations, so-called "dust granuloma," characterized by cellular inflammatory reaction with epithelioid cells, fibroblasts, and plasma cells. He was unable to produce any fibrosis or anything similar to a pneumoconiotic nodule, though granulomas due to barite did appear and gave a roentgenographic picture similar to the pneumoconiotic nodule. He had used 1 cc. of 5 per cent or 10 per cent suspension for a guinea pig. He remarked that rabbits and guinea pigs tolerated the injection well, even of large quantities or in high percentage of barite. He used 3.5 cc. for a rabbit. He mentioned that there was some mortality, but did not specify how high it was in the experimental animals.

In 1934, Singer[93] reported 4 cases having perforation of a peptic ulcer following a roentgen examination of the upper gastro-

intestinal tract, but did not discuss specifically the reaction of barium on tissue. Sullivan[96] in the same year reported a single male patient, aged seventy, who, in a weak condition, aspirated "a fair quantity of the barium" and died a few hours later. No postmortem examination was done.

In 1949 Crandall[26] reported an investigation of the absorption of barium sulfate from the gastrointestinal tract of rats, using radioactive isotopes. He showed that the barium sulfate, which is slightly soluble at body temperature (0.25 mg. per 100 cc. water) is absorbed by the digestive tract and can be demonstrated throughout the body in very small amounts. This experiment appears to be important in showing that barium is not totally "inert" physiologically, although it has never been shown to be actually toxic when administered via the gastrointestinal route. The very slight solubility of barium sulfate may help to explain its effect on the lung parenchyma.

In 1950 Brown *et al.*[19] reported an investigation of particle size upon retention of particulate matter in the human lungs. Using nebulized barium sulfate and analyzing its content in inspired and expired air, they determined that a particle size of approximately 1 micron is optimum for alveolar retention of inhaled dust. In this connection U.S.P. barium sulfate as supplied commercially has been reported by Adolph and Taplin[1] as 97 per cent (by weight), 5 to 20 micron diameter particle size, and later by Shufflebarger *et al.*[91] as around 1 to 1.5 microns in size. This would indicate that the commercially supplied barium preparation in routine roentgenologic use is close to the optimum size for deep penetration of the lung when injected in suspension into the bronchial tree.

In 1952 Roman *et al.*[82] described a case of massive barium embolism during the course of an examination of the colon when an obturating balloon catheter was being used. Death followed almost immediately and the opaque medium was identified in the abdominal, renal and pulmonary vessels. They cited the only previous case of barium embolism as that of Isaacs *et al.*[52] in 1950.

Kleinsasser and Warshaw,[58] also in 1952, reported a case of perforation of the sigmoid colon during a barium enema examination, with recovery of the patient after a stormy course. They injected 20 to 150 cc. of barium into the peritoneal cavities of 6 dogs. All survived except one which died two months later of other causes. No significant adhesions were found in any of the animals. They stated that "as in all other studies done, the typical finding was of barium granuloma on the surface of the gut and peritoneum." Early changes included inflammation, edema and congestion; later only patchy areas of chronic inflammation and barium granulomata appeared.

Huston *et al.*[51] in 1952 experimented on rats to determine the effect of barium sulfate on the lungs. They injected the opaque medium through the larynx into the trachea by a special pharyngeal cannula which temporarily occluded the larynx while the injection was made. Roentgenograms were taken immediately before and again after injection. The animals were sacrificed at time intervals from thirty minutes to one hundred and twenty-six days. Autopsies were performed immediately and sections of the lungs examined microscopically. They found four more or less clearly defined patterns: (1) An initial inflammatory reaction with large numbers of polymorphonuclear cells. There was little evidence of fluid. The changes were seen at twelve hours; (2) a mononuclear infiltrate appearing from forty-eight hours to fifteen days. Areas of consolidation were filled with barium sulfate; (3) a stage of mononuclear disintegration at around thirty days; and (4) a stage of tissue reaction at ninety-four to one hundred and twenty days. The lungs for the most part, however, appeared normal, and there was no suggestion of fibrosis. Refractile masses with a few lymphocytes and mononuclears were present. They suggested that once barium sulfate enters the lung substance its

removal by mononuclear transportation is in some way inhibited. Once the cells had ingested barium they appeared inert. The barium sulfate could exert a toxic effect producing degenerative changes in polymorphonuclear and mononuclear cells and on the respiratory epithelium. They concluded that barium sulfate is a relatively inert substance in tissues and gives rise to only a very mild reaction. The barium cannot be completely removed from the lungs.

Cember *et al.*[23] in 1954 investigated the ability of particles of barium to penetrate the lungs when administered intratracheally to rats. Particulate penetration and distribution were determined with the aid of autoradiographs. Retention of particles was determined by direct measurement of activity in the lungs. The mean particle size was 1.45 mμ. (This dimension had previously been shown to be in the optimum range for deep pulmonary retention.) They used 90 rats and injected very small amounts of barium sulfate suspended in distilled water and tagged with radioactive sulphur. They showed that the particles were dispersed in all the lobes of the lung, and could be found in irregularly shaped areas throughout all parts. About 40 per cent of the materials injected into the trachea was deposited in the deep respiratory tract. There was an initial rapid elimination of the dust particles by direct transport up the ciliated epithelium in about six hours. The second, slower part of the elimination was thought to represent removal from the alveolar sacks by phagocytosis. The half life of the barium in the alveolar sacks was calculated to be about 3.3 days. Their experiments, however, were dissimilar from those reported here in that the amounts of barium injected were very small, presumably too small to produce shadows on roentgenograms.

Kay,[56] in 1954 reporting 2 cases of intestinal perforation with barium entering the peritoneal cavity and one case of a tumor in the wall of the rectum following barium enemas, stated that barium sulfate produces definite but minimal fibrous tissue and inflammatory response. The next year Kay and Choy[57] reported experimental observations following the intraperitoneal injection of large doses of sterile barium suspension in mice. The basic response to the injected medium was granuloma formation, later causing fibrous adhesions. The ultimate effects, however, did not appear to be deleterious.

Fite[39] in 1955 had a case of "barium granuloma of the lung following accidental aspiration of radiographic contrast medium." In this case the material aspirated was gastropaque, containing a pentosan and vanilla flavoring as well as barium sulfate. Five rabbits were injected endotracheally with gastropaque and when killed fifteen days after three daily injections had been made, showed a similar reaction to that found at autopsy in his patient. Fite did not establish whether the reaction was due to the barium, or to the ingredients of gastropaque.

Krizan *et al.*[59] of Bratislava reported in 1955 on their experience with a watery suspension of barium sulfate, to which mucin had been added to achieve the desirable viscosity. The barium used was a 35 per cent suspension. When dogs were injected with low viscosity barium suspension, histologic examination showed atelectasis and venostasis, without evidence of inflammation. With adequate viscosity, the filling material was shown to adhere to the bronchial wall. It gave a sharp outline of the bronchial tree. When the contrast material did not fill the alveoli, it was coughed up within twenty-four to forty-eight hours. These experiments on dogs under anesthesia were felt to show that the barium-mucin mixture was adhesive and highly opaque roentgenographically and completely innocuous. It therefore appeared likely that the material would prove suitable for human use, providing it could be shown that it was not irritating to the bronchial tree.

Šári *et al.*[84] in 1956 used Krizan's barium suspension in humans. They tried solutions of different consistency, adding mucin or

bentonit or both to arrive at the proper viscosity. Twenty-three bronchographies were done on 21 patients. The examinations were reported as well tolerated in almost all cases and causing less irritation than with other radiopaque material. There was good adhesion of the radiopaque material to the bronchial mucosa and excellent roentgenograms could be obtained (we have been unable to obtain the original article and our photostatic copy of it did not permit study of the reproductions). They stated that 6 cases showed filling of the alveoli, but, in all, the radiopaque material was expectorated and only minimal amounts remained in the alveoli. They felt that further experience with material of the right consistency would enable them to avoid alveolar filling. They concluded that the medium was harmless and valuable for bronchographic examinations in man. No pathologic examination was reported.

Di Rienzo and Pereira-Duarte[30,31] in 1956 and 1957 reported enthusiastically on the use of barium sulfate suspended in water with added CMC. They stated that the barium sulfate is retained sufficiently long to allow leisurely study of small and middle sized branches of the bronchi but is eliminated by coughing in two or three days after the procedure. They feel that there is no danger of finding traces of the opaque medium in the lungs months after the bronchography as occasionally happens with lipiodol and say that toxic side effects do not occur. They recommend it for routine use in bronchography. No pathologic examination is reported by them of human or animal material.

DISCUSSION

The purpose of the present investigation has been primarily to determine what opaque medium, if any, combines the features of usefulness and safety when it is likely to be aspirated into the respiratory tract incidentally to the examination, rather than the choice of the most suitable medium for bronchography. It is evident, however, from the recorded literature that most of the information available in regard to the effect of the various media on the respiratory tract has been derived from investigations orientated toward bronchography. The opaque media available may be briefly summarized as follows:

Bismuth and cesium do not appear to have proved their worth and have been largely abandoned. Umbradil viskos and joduron B do not appear suitable because of high viscosity and irritation of bronchial mucosa.

Lipiodol is not without danger when aspirated in the lung, is retained in the pulmonary parenchyma for prolonged periods, and, as long as it is retained, interferes with roentgenologic visualization of the lungs. In addition, it has been shown to enter the trachea and bronchi when ingested or even injected into the nose or throat of normal individuals[66] and therefore may give a false positive diagnosis of tracheo-esophageal communication (Fig. 10 and 11). DeCarlo *et al.*[29] showed that, when lipiodol was given by mouth to normal infants, 13 per cent of the infants aspirated the oil into the tracheobronchial tree. The subjects who showed this phenomenon with the oily opaque medium did not aspirate barium sulfate when it was fed to them under similar conditions

Dionosil aqueous is mildly irritating in the upper respiratory tract, and contains carboxymethylcellulose, which may be injurious to the lungs if aspirated, though the effect of CMC on lungs is still controversial. In addition, its high viscosity does not make for ease of handling in the investigation of the pharynx and esophagus of patients with swallowing disorders.

Dionosil oily contains arachis oil. It therefore has some of the disadvantages of lipiodol except that it does not cause a persisting lung radiopacity.

Lipiodol with added sulfonamide (visciodol) appears to have some advantages for bronchography, but is not likely to be suitable for administration by mouth in the

investigation of swallowing disorders in view of its oily nature and high viscosity.

Barium sulfate has the advantages of ease of handling and availability and high radiopacity; it is not liable to enter the respiratory tract in the majority of normal individuals, during the act of swallowing (but see Fig. 12). However, its retention in body tissues, including the lung has been reported to be injurious, though not markedly so. No direct comparison of barium sulfate with the other opaque media in lungs or bronchi has been made previous to the present investigation. It therefore appears that there was no basis for decision as to its usefulness or otherwise in patients with swallowing disorders.

Our investigation has two obvious weaknesses.

1. The results of animal investigations have only limited application in work with humans. There can be no question about the validity of this objection, but it is only by animal investigations that objective and controlled observations of toxicity and tissue damage can be made. It may be that similar controlled experiments should be carried out in other species to determine whether our findings are more generally applicable.

2. The opaque media were injected in these experiments almost directly into distal bronchi and alveoli in many animals rather than into the larger bronchi. This was inherent in the method, which required blind insertion of a small polyethylene catheter into the bronchus until it was arrested. This technique, however, was the same in all animals with the opaque media and with the tap water injections. It may be argued that with adequate viscosity (as in lipiodol with sulfonamide and barium with CMC) the opaque medium should not reach the alveoli in appreciable quantities in bronchography. In the investigation of swallowing disorders, however, high viscosity and oily mixtures are not altogether desirable and low viscosity suspensions, if aspirated, may well reach the alveoli, as shown in our case. It is therefore desirable to determine as far as possible their effect on the lung parenchyma, as well as the bronchial tree.

The large amounts of alveolar flooding in our animals could be expected to produce maximum damage in the segments of the lung injected.

The use of the stain for lipiodol described by Felton appeared to be sufficiently satisfactory in the rat lungs to be of value in identifying the same substance in the lung of our human case. It was therefore established with reasonable certainty that both opaque media had been retained in this infant's right lung, but it was not established by the histologic appearance whether one was present in larger amounts than the other or had produced lung damage.

It should be emphasized that the method used in our animal experiments was a comparative or quantitative, rather than a qualitative one. In grading the results from histologic observations and from roentgenograms, we were attempting to learn whether a consistent difference could be established in the early and late tissue reactions, damage to lung parenchyma, and retention or elimination of the injected substances. Much has been done by others in determining the role of the various tissue cells in the absorption, elimination or retention of foreign material in the lung, and though much more remains to be done, our study was not intended to contribute to these problems.

We did not feel that the early and late death rates in our series were highly significant. The slightly higher mortality in the lipiodol injected animals was possibly related, however, to the viscosity of the oily medium, which could be observed to cause obstruction of the very small tracheal lumen of the rat rather easily. This observation is of limited value, but may suggest that high viscosity media, while valuable in preventing alveolar flooding, may in themselves be capable of causing a significant degree of obstruction of small bronchi.

The differences in histologic changes produced by the various media were more marked in the early than the late stages. Thus the barium in sucrose caused the most marked early edema and inflammatory cell response, dionosil the next, and lipiodol slightly less. Barium in tap water produced a moderate early cellular reaction, but little edema. The late histologic effects were not severe in any group. Lipiodol seemed to produce the most marked late effects, barium in sucrose solution the next, barium in tap water a little less, and dionosil the least. Barium was clearly retained in the lung much longer than any of the other opaque substances, and lipiodol, next longest.

Although the sucrose solution in which the barium was administered was approximately isotonic, there appeared to be no doubt that its effect on the lungs was worse than that of barium in tap water.

CONCLUSIONS

It was not established that of the opaque media tested any one is clearly contraindicated in the investigation of swallowing disorders on the basis of its possible effect on the lungs.

We have used a suspension of barium in sucrose to feed our infants during gastrointestinal problems; the sugar should not be used in the examination of patients with swallowing disorders. It would appear that an investigation of barium in tap water as compared with barium in normal saline might be warranted, but we have not made such a study.

Our results suggest that although the initial reaction is considerable with dionosil aqueous, its late effect is less than with barium or lipiodol.

In view of the inability of some normal infants to swallow oily substance without aspirating it, the following method of investigating swallowing disorders, at least in infants, is suggested:

A nasogastric catheter is inserted into the lower esophagus, then the mid-esophagus, then the upper esophagus, and at each level dionosil aqueous is injected. Sufficient pressure is used during the injection to slightly distend the esophagus. This is done with the patient lying on his side in lateral projection under fluoroscopic control. Spot roentgenograms are made during the procedure, whether or not any opaque medium is seen to enter the trachea. If no opaque medium enters the trachea, the catheter is withdrawn and barium in water (or saline) is given by mouth. Its passage through the mouth, pharynx and esophagus is studied and its entry into the trachea, if it occurs, is observed and recorded by spot roentgenograms. This method appears to permit the identification and localization of a tracheo-esophageal fistula.

SUMMARY

An investigation has been carried out in rats to determine the effect on the tracheobronchial tree of injected opaque media as compared with water.

Of the opaque media investigated, including lipiodol, aqueous dionosil and barium sulfate, no one appears to be clearly more damaging to lung parenchyma than the others.

A method of investigation of swallowing disorders in infants is suggested.

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Representative on the Board of Chancellors of the American College of Radiology: Charles L. Martin, Dallas, Texas.

Forty-second Annual Meeting: The Caribe Hilton, San Juan, Puerto Rico, March 17–19, 1960.

EDITORIALS

SIXTIETH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Sixtieth Annual Meeting of the American Roentgen Ray Society, the oldest scientific radiologic organization in the United States, was held at the Netherland Hilton Hotel, Cincinnati, Ohio, Tuesday, September 22, through Friday, September 25, 1959.

After the call to order by Doctor Barton R. Young, President of the Society, the Address of Welcome was given by Mr. Walter C. Langsam, President of the University of Cincinnati. This was a very pleasant prologue to the beginning exercises of the Society's sixtieth meeting.

Immediately following the Address of Welcome, Doctor Edward B. D. Neuhauser was installed as President. The subject of his Inaugural Address was "Planning the Program." This address is published elsewhere in the JOURNAL. If any proof was needed as to Doctor Neuhauser's capabilities in "planning the program," one only had to attend the Scientific Sessions of the Society which began immediately after his presidential address.

The first five papers of the Tuesday morning session were devoted to cranial and intracranial lesions. This very instructive group of papers was followed by one dealing with *Neurogenic Tumors of the Mediastinum—A Clinical Pathological Study.*

Doctor Joseph E. Whitley and Doctor H. Francis Forsyth, Winston Salem, North Carolina, then gave an exceedingly interesting paper on *The Classification of Cervical Spine Injuries.* They discussed in summary the findings in 137 cases they had analyzed, with classification based on the history, roentgenographic, clinical and surgical findings.

A paper on *The Radiological Diagnosis of Mesentericoparietal Herniations* followed. This paper discussed the formation of the various fossae of the posterior parietal peritoneum in the vicinity of the duodenum through which the herniations might occur. The differential characteristics of three types of hernias were demonstrated. This paper was very important because attention was called to the part the radiologic study might play in recognizing the abnormal pattern and assessing the clinical data.

The final paper of the Tuesday morning Scientific Session dealt with *Ovarian Irradiation and the True Hormonal Menopause.*

The afternoon sessions of the Society were devoted to a study of the Scientific and Technical Exhibits, and to the Instructional Courses.

Doctor Harold O. Peterson, who is Director of the Instructional Courses, had as Associate Director, Doctor Donn G. Mosser of Minneapolis, Minnesota. They had assembled a splendid faculty of instructors who covered all phases of radiology in their Instructional Courses—namely, diagnostic roentgenology, therapeutic radiology, nuclear medicine, physics and radiobiology. The Instructional Courses have assumed a place of great importance at the Annual Meetings of the Society, and under the directorship of Doctor Peterson, they have grown not only in popularity but in the variety and quality of instruction offered.

As is customary, the Annual Caldwell Lecture was given Tuesday evening. This year's lecture was given in the Pavillon Caprice by Doctor Donald L. McRae, Associate Professor of Radiology, McGill University, and Radiologist-in-Chief, Montreal Neurological Institute, Montreal, Canada.

Doctor McRae was introduced by Doctor Carleton B. Peirce, Professor of Radiology, McGill University, and Radiologist-in-Chief, Royal Victoria Hospital, Montreal, Canada. Doctor McRae chose for the topic of the Caldwell Lecture, *The Significance of Abnormalities of the Cervical Spine*. On completion of the Lecture, Doctor McRae was awarded the Caldwell Medal, which signifies one of the highest honors bestowed by the Society.

The Scientific Session on Wednesday morning continued with a group of papers of great interest in varied subjects, including papers dealing with radiation accidents and cardiovascular anomalies.

Several papers dealing with various types of pneumonia proved to be of considerable interest because of the apparent rarity of some of these pneumonias, including giant cell pneumonia, interstitial plasma cell pneumonia, and pneumocystis Carini pneumonia, which the essayists believe is becoming more prevalent.

An unusual paper on *Renal Tubular Ectasia in Cystic Disease of the Kidneys and Liver* was given by Doctor Bernard J. Reilly, Saskatoon, Saskatchewan, Canada, in association with Doctor Edward B. D. Neuhauser, Boston, Massachusetts. It was noted by the authors that a few sporadic cases had occurred at the Children's Hospital in Boston during the past twenty years, but it was the discovery of a family carrying the heredofamilial trait of cystic disease which precipitated the present investigation.

The next paper was one on *Some Observations on Gastric Disease in Pediatric Radiology*. A summary was given by the author, Doctor Harvey A. Humphrey, St. Louis, Missouri, of his personal experience with congenital and acquired lesions including instances of duplications, leiomyomas, Menetrier's syndrome, volvulus, malrotation, and peptic ulcer.

A discussion on *Increased Intracranial Pressure—Roentgen Studies in Infants and Children* immediately followed the above paper.

An Assessment of the Value of Dehydration in the Preparation for Intravenous Pyelography was also presented. The authors called attention to the fact that it has been generally accepted that dehydration is necessary in the preparation of all patients for intravenous pyelography, although one investigator stated that "In spite of rigid dehydration we were unable to obtain successful pyelograms in these children." The problem was authoritatively discussed.

On Thursday morning, the first paper described a relatively new device for *Rapid Automatic X-ray Film Processing*. The paper was based on the personal experience of Doctor Everett L. Pirkey, Louisville, Kentucky. He gave a brief discussion on the use and great help of rapid film processing.

A group of papers dealing with cineradiography followed. These papers called attention to the added information which may be obtained from this type of study in pediatric radiology, particularly in the upper gastrointestinal tract, and one paper discussed the dosimetry and some other physical factors relating to serial and cineradiography.

Doctor C. C. Wang and Doctor John D. Reeves then presented a paper on the *Roentgen Manifestations of Mesenteric Vascular Disease*.

Serial Femoral Arteriography in Occlusive Disease: Clinical-Roentgenologic Considerations was a presentation concerning an evaluation of approximately 100 femoral arteriograms. Clinical, operative and roentgenologic correlations were made in this group of cases. These arteriograms were performed with a newly designed automatic long-segment serialograph in use since October, 1957. The operation of the serialograph was described and illustrated, and the advantages of such a unit were emphasized.

A discussion on the *Segmental Dilatation of the Colon during the Course of Fulminating Ulcerative Colitis: Roentgen Findings* was given by Doctors Bernard S. Wolf and Richard H. Marshak, New York City.

They pointed out the importance of realizing that a simple roentgenogram of the abdomen in this condition shows characteristic findings in almost all instances. Barium enema examination is unnecessary and undesirable. This report outlined their experience with 16 patients, 9 of whom required emergency surgery, with 3 deaths—1 postoperative. The findings as seen on the simple roentgenogram were discussed in detail.

Familial Recurring Polyserositis: Clinical and Roentgen Diagnosis was the subject of a discussion by Doctors William R. Eyler, Robert K. Nixon and Robert J. Priest, Detroit, Michigan. The authors have observed 20 patients with this disorder. In 10 patients studied during episodes of abdominalgia, the small intestinal motor activity was abnormal. Pleural attacks resulted in a transient pleural effusion or basilar atelectasis. These nonspecific roentgen findings have been helpful in establishing the diagnosis.

Doctor Maurice D. Sachs and Doctor Philip Partington, Cleveland, Ohio, presented a paper dealing with *Distended Gallbladder: The Value of a Fat Meal in Cholecystography*.

Doctor Harry W. Fisher of Iowa City, Iowa, discussed problems concerning *The Big Duodenum*.

The concluding paper on Thursday morning was *Osteoblastic Metastases in Bronchial and Intestinal Carcinoid*, given by Doctors Frances Toomey and Benjamin Felson, Cincinnati, Ohio.

On Thursday evening the Annual Banquet was held in the Pavillon Caprice, a delightful place. The President of the Society and his Program Committee this year deviated considerably from the routine in that Doctor Emmett O'Grady, Professor of English and Head of the English Department, University of Ottawa, was selected as speaker of the evening. He gave a very witty and learned discourse on *Levity and Learning in Literature*. During the evening the new officers of the Society were also introduced and there was a presentation of the awards of the Scientific Exhibits. The banquet was followed by dancing.

The Scientific Session on Friday morning was devoted to papers dealing with various phases of radiation therapy, interesting problems of radiation research and some miscellaneous topics. Among the therapy papers were: *Cobalt 60 Radiography*, used not only for localization of therapy ports but also for the demonstration of soft tissue lesions in the chest, neck, and nasopharynx, which are not visible on routine roentgenograms; *The Design of a Second Cobalt 60 Unit Based on Experience of 1,000 Cases Treated with the First Unit*, as practiced at the M. D. Anderson Hospital, Houston, Texas; *Radiation Therapy of Benign Conditions*, expounding the indications and contraindications in this type of lesions, particularly in the light of the confusing concepts of recent years; *Lymphosarcoma: Long Term Results following Radiation Therapy*, which gave statistical evaluation of the results in 514 cases treated since 1922; and *Preliminary Experience with Split-Dose or Periodic Radiotherapy*, in which the author discussed the use of discontinuous, split-dose or periodic irradiation as contrasted to accepted conventional radiotherapeutic methods.

The research papers included: *Response and Recovery of Normal and Grafted Skin to Ionizing Radiation; Contribution of Plasma Constituents Produced following Whole Body Irradiation on the Efficacy of Local Tumor Radiotherapy*, and *The Effect of Triiodothyronine on Radiosensitivity*.

In the miscellaneous group, there was an interesting paper on physics, *Isodose Curves about the Beds of Patients Containing Radium and/or Cobalt 60*, in which the author stated that these curves did not agree with the anticipated curves plotted from the data in the Handbook of the National Bureau of Standards because of relative "hot spots" presumably from secondary scattering from the bed and the patient; and a paper on *A Critical Appraisal of the Diagnostic and Prognostic Value of Radioiron Study in Hematologic Disorders*, in

which the physiology of iron and hemoglobin metabolism was discussed and the tracer data of the radioiron turnover in a number of anemias were presented.

On Wednesday afternoon, preceding the Instructional Courses, the motion picture *Radiation: Physician and Patient* was shown. This motion picture brings together for the first time in one setting the nation's most authoritative scientists and physicians on radiation protection and hazards. It was sponsored by the American College of Radiology in cooperation with the U. S. Public Health Service on a grant from the Rockefeller Foundation. The 16 mm. color film is about forty-five minutes in length and presents the use of radiation in its entire medical framework. Doctor Wendell G. Scott, former President of the American Roentgen Ray Society, Professor of Clinical Radiology, Washington University School of Medicine, St. Louis, Missouri, was Chairman of the committee of the American College of Radiology which was responsible for production of the film.

Doctor Scott in his introductory remarks before the presentation of this excellent motion picture stated that additional prints of the film will be made available without charge for professional audiences, medical society meetings, hospital staff conferences and other groups whose interest lies in the use of medical radiation. These prints may be obtained from the American College of Radiology, the American Medical Association and the United States Public Health Service.

The motion picture *Radiation: Physician and Patient* represents another important milestone in the continuing education program of the American College of Radiology in radiation protection among the nation's physicians.

The Scientific Sessions of the Society were preceded on Monday by the Annual Golf Tournament for members and guests. Arrangements for this tournament were made by Doctor Harold G. Reineke and his Committee on Local Arrangements, and the tournament was held at the Kenwood Country Club. This tournament is an annual event of the Society for the Willis F. Manges Trophy which is awarded for the Eighteen Hole Medal Handicap for members, and for the Exhibitors' Trophy which is awarded for the Eighteen Hole Gross Score for members. The Annual Golf Dinner was also held at the Kenwood Country Club.

Doctor Reineke and his Committee on Local Arrangements deserve the thanks of the Society for not only arranging the Golf Tournament but for contributing greatly to the success of the meeting in helping with the various details.

Under the Chairmanship of Mrs. Harold G. Reineke, the Ladies' Committee provided an excellent program for the wives of members and guests. To this Committee the Society expresses its thanks.

For the Manager and staff of the Netherland Hilton Hotel the Society has nothing but profound appreciation for the splendid manner in which all details of the meeting were carried out and for the courteous treatment accorded members and guests by its staff.

The Scientific Exhibits were numerous and most instructive. A detailed report of these is given elsewhere in this issue of the JOURNAL.

The Technical Exhibitors arranged a magnificent display of the various types of technical equipment used by radiologists, and the Society is extremely grateful to them for their contribution to the success of the meeting. The list of the exhibiting firms is given elsewhere in the JOURNAL.

The newspaper publicity of the Program was conducted under the able directorship of Mr. Hugh H. Jones, a member of the Executive Staff of the American College of Radiology.

It may be stated that the Sixtieth Annual Meeting of the American Roentgen Ray Society was excellent in every respect and one which long will be remembered.

PATRICK F. BUTLER
1877-1959

IN THE death of Dr. Patrick F. "Paul" Butler on June 29, 1959, the medical profession and the community lost an exemplary member. Born in Boston, on June 13, 1877, he received his pre-medical education at the College of the Holy Cross and Harvard College. Following his graduation from Harvard Medical School in 1903, he served for two years as surgical intern at the Carney Hospital, then devoted several years to general practice before entering the then new field of roentgenology. He re-

ceived his initial training under two of the country's pioneer roentgenologists, Doctors Percy Brown and Francis Williams, later succeeding Dr. Williams as Chief of the Department of Roentgenology at the Boston City Hospital. Subsequently Dr. Butler entered the private practice of roentgenology in Boston, and continued active in this work until a few months preceding his death.

Dr. Bulter lived a rich and full life marked by many outstanding professional accomplishments. An early achievement was the establishment of the x-ray department in Saint Elizabeth's Hospital, one of Boston's largest Catholic hospitals. Beginning in 1922, however, he devoted his hospital work entirely to the Boston City Hospital where he remained as Chief until his resignation in 1945. He continued to serve as Consultant in Radiology at the Boston City Hospital, Carney Hospital, Saint Elizabeth's Hospital, and Beth Israel Hospital.

In his work with residents and medical students in the field of roentgenology, he was a most skillful and devoted teacher who possessed the gift of communicating spiritual and professional enrichment along with technical proficiency. The more than fifty graduates of his resident training program, now practicing in almost every state, not only received expert preparation in this specialty but were also inspired to develop their senses of humaneness, charity, and practical medicine. Medical students benefited likewise from his rare spirit of dedication during the many years he was Clinical Professor of Radiology at Harvard Medical School.

Despite the demands of his hospital duties, his busy private practice, and his teaching and training programs, Dr. Butler's professional zeal was such that he engaged in many other professional activities. He was the author of more than a dozen scientific papers and he gave freely of his time to lecture to lay and legal groups on many facets of his specialty. He was a member of the Massachusetts Medical Society, American Medical Association, Radiological Society of North America, and the American College of Radiology. He was also a member and Past President of the American Roentgen Ray Society and the New England Roentgen Ray Society.

In recognition of his many contributions to the branch of medicine in which he was pre-eminent, Dr. Butler recently received two honors which he especially cherished. In 1957 the College of the Holy Cross, where he received his early pre-medical training, conferred on him the honorary degree of Doctor of Science; and in 1958 he was chosen by the New England Roentgen Society to give the George Holmes Lecture, named for an old and valued friend.

Surviving Dr. Butler are his sister, Mrs. May Ferguson, and a brother, Frank. Dr. Butler's many friends and associates join in offering condolence to the family of this humble man who was the essence of everything fine in a doctor, a humanitarian, and a friend.

Albert M. Moloney, M.D.

We regret to announce the death on September 10, 1959 of Dr. Simeon T. Cantril, a distinguished member of the American Radium Society.

THE SCIENTIFIC EXHIBITS

THE Scientific Exhibits of the Sixtieth Annual Meeting of the American Roentgen Ray Society, held at the Netherland Hilton Hotel, Cincinnati, Ohio, September 22–25, 1959, were of an unusually high standard and contributed notably to the success of the entire meeting. President-Elect Dr. Edward B. D. Neuhauser and the Scientific Exhibits Committee, under the Chairmanship of Dr. Richard H. Marshak, spent considerable time in making the necessary arrangements, which proved so fruitful. Their efforts were amply rewarded by the great interest shown by the many members and guests of the Society who visited the Exhibits. The display was splendid in its ensemble and the individual contributions comprised some of the most important achievements in present-day scientific radiology. The Committee on Awards after careful consideration, awarded one Silver Medal, two Bronze Medals and seven Certificates of Merit.

Following are brief descriptions of the exhibits.

First Award: Silver Medal. *Response and Recovery of Normal and Grafted Skin to Ionizing Radiation.* Philip Rubin, M.D., Capt. Jerry Grise, MC, USAF, George Casarett, Ph.D., Lester Cramer, M.D., and Anatol Ryplansky, M.D., University of Rochester, Strong Memorial Hospital, Rochester, New York.

A definite difference in response of normal and grafted skin to irradiation has been noted clinically in a variety of settings. Since reconstructive surgery is increasing with the advent of more radical operative procedures for various carcinomas, consideration of postoperative irradiation in grafted sites is not unusual.

Detailed experimental studies were presented which illustrated three categories of graft age and radiosensitivity. In new grafts less than three months old, irradiation demonstrated greater sensitivity of the grafted sites than in normal skin. In a graft of intermediary age, the response and recovery to irradiation paralleled that of normal skin. In several grafts older than one year, no reaction to irradiation was elicited, but in one instance, graft necrosis ensued at the sixteenth week.

On the basis of the results obtained, the following suggestions worthy of consideration in the irradiation of grafts were made: (1) limit the total dosage to a graft treatment area as much as possible; (2) exclude as much of the graft as possible from the radiation portal such as is consistent with good treatment; (3) fractionate and protract the dose since better recovery is insured; (4) employ supervoltage irradiation because the skin sparing effects thereby lessen the severity of the reaction; and (5) allow for a good "take" of the autograft rather than starting irradiation immediately.

The differences in the vascularization of normal skin and grafted skin form a reasonable basis to explain the differences in radiation reactions of these structures. Careful clinical observations and experimentation have clearly shown that in a wide variety of circumstances alteration in capillary anatomy and physiology has a profound effect on irradiated tissue and tumors. Knowledge of the changing blood supply pattern of an autograft, therefore, is essential. The stages in graft union to host tissues are those of (a) plasmic circulation, (b) vascularization, (c) organic union, and (d) cicatrization. It is logical to anticipate that the more vascularized a graft is, the more sensitive the graft will be to irradiation.

Second Award: Bronze Medal. *The Sphincteric Function of the Uterine Isthmus.* Edward C. Mann, M.D., William McLarn, M.D., and David B. Hayt, M.D., The New York Hospital—Cornell Medical College, New York, New York.

Recent research indicates that the uterine isthmus performs a sphincteric function throughout most of pregnancy.

By means of a two-stage intrauterine

balloon, this sphincteric function, or the lack of it, was studied roentgenographically in the nonpregnant state. Conventional roentgenographic and cinefluorographic techniques were employed.

To date, balloon studies have been performed upon 350 habitual aborters and upon 100 nonaborting controls. The roentgen findings coupled with clinical follow-up clearly revealed that cervical incompetence can now be diagnosed in the nonpregnant state and that the sphincteric disability resides in the isthmus rather than the cervix.

Third Award: Bronze Medal. *Experimental Intraocular Venography*. Bruce E. Cohan, M.D., and Joseph M. Morel, R.T., Ophthalmology Branch, NINDB, National Institutes of Health, Bethesda, Maryland.

Experimental intraocular venography is a new technique which permits the study of the intraocular venous systems in the living intact cat eye. The intricate venous complexes within the sclera, ciliary body and choroid, inaccessible to the most advanced clinical methods, can be visualized by this technique.

A study of the dynamic physiologic anatomy of these vessels, now possible by means of intraocular venography, promises to help solve problems of etiology and diagnosis in the glaucomas and in inflammatory heredodegenerative, geriatric, and neoplastic diseases of the eye.

The angiographic method used in the cat's eye was demonstrated. It can be employed equally as well in the dog's eye. Application in man awaits further modification of cannulation technique.

Certificates of Merit

1. *Total Body Irradiation and Tissue Transplantation*. James B. Dealy, Jr., M.D., Richard E. Wilson, M.D., Fred Lee, M.D., and Norman L. Sadowsky, M.D., Departments of Radiology and Surgery, Harvard Medical School and Peter Bent Brigham Hospital, Boston, Massachusetts.

The use of total body irradiation to alter immunogenetic specificity has become an established procedure in laboratory animals. This exhibit demonstrated the ability of irradiation to induce a form of acquired tolerance to single donor and pooled homologous bone marrow and to homologous skin in the rabbit.

Some practical and theoretical human applications were considered.

The beautiful white rabbit, used in the demonstration throughout the meeting, was awarded a blue ribbon.

2. *Gas Tubes and Glass Plates*. John M. Dennis, M.D., and James J. Stovin, M.D., University of Maryland School of Medicine and University Hospital, Baltimore, Maryland.

This exhibit included old roentgen-ray tubes which depicted the evolution of the modern tube, glass plates, and other museum items of interest to radiologists.

The following tubes were displayed: (1) Puluj tube; (2, 3) Jackson type tubes; (4) R-F standard tube; (5) Platinum target tube with osmoregulator of P. Villard; (6) Gundelach-Dessauer tube; (7) Platinum-nickel-copper anticathode tube; (8) Double target tube; (9) Queen self regulating tube; (10) Valve tube; (11) American tube with carbon softening device; (12) Macalaster, Wiggin Company tube; (13) G. & E. tube; (14) Mammoth air cooled tube; (15–19) Macalaster, Wiggin Company tubes; (20) Water cooled French tube with Bauer regulator; (21) Universal Coolidge tube; (22) Radiator Coolidge tube; (23) Portable unit tube; (24) Line focus tube; (25) Modern tube; and (26) Rotating anode tube.

Mimeographed descriptions of each tube with a brief chronologic résumé of their evolution were supplied.

3. *Tomography in the Diagnosis of Carcinoma of the Paranasal Sinuses*. Robert L. Egan, M.D., John E. Healey, M.D., and Gerald D. Dodd, M.D., M. D. Anderson Hospital and Tumor Institute, Houston, Texas.

Experience with over 200 patients with primary or secondary carcinoma of the paranasal sinuses has led to great dependence upon laminagraphic studies for diag-

nosis and treatment. The correlation of the normal anatomy with the laminagrams of this area was depicted and cases illustrating the value of this type of study were presented.

4. *Vertebral Fractures with Demineralization; A Study of 105 Patients.* Robert H. Freiberger, M.D., James A. Nicholas, M.D., and Philip D. Wilson, M.D., Hospital for Special Surgery, Cornell University Medical Center, New York, New York.

This exhibit consisted of statistical data of 105 patients admitted to the hospital with bone demineralization and compression fractures of vertebrae not caused by severe trauma or known malignant tumor. Criteria for the roentgen diagnosis of bone demineralization were discussed.

5. *Contributions of Plasma Constituents Produced Following Whole Body Irradiation on the Efficacy of Local Tumor Radiotherapy.* James E. Lofstrom, M.D., and Melvin R. Sikov, Ph.D., Wayne State University College of Medicine, Detroit, Michigan.

This exhibit contained graphs, charts and descriptive material depicting the results of radiobiologic research on the contributions of whole body irradiation to the efficacy of local tumor therapy in various experimental tumors in mice;—*i.e.*, Krebs carcinoma, Ehrlich carcinoma, C_3HBA adenocarcinoma, sarcoma 37, L_1 lymphosarcoma and H-P melanoma. Also results of injected pre-irradiated plasma were shown. A definite but variable pattern was observed.

6. *Routine Air-Contrast Examination of the Duodenal Bulb.* George A. Moller, M.D., and John L. Williams, M.D., Geisinger Memorial Hospital and Foss Clinic, Danville, Pennsylvania.

Air contrast study of the duodenal bulb is of primary value when the bulb cannot be compressed or palpated adequately. A significant number of ulcer craters can be discovered by this technique which are not demonstrable on conventional roentgenograms.

This technique is recommended in routine examination of the upper gastrointestinal tract. It consists of an initial routine study in the erect position, including pressure spot roentgenograms of the duodenal bulb. The patient is then given 1 ounce of a carbonated beverage to ensure an adequate amount of gas in the stomach. He is placed in the supine position and rolled toward his left side so that the gas rises into the distal antrum and enters the duodenal bulb. Spot roentgenograms of the air-filled bulb are obtained. The study of the stomach and duodenum is then completed with fluoroscopy and roentgenograms in the prone-oblique position.

7. *Subcutaneous Lymphangiography in the Human.* J. M. Protas, M.D., and R. N. Cooley, M.D., Radiology Department, and J. R. Smith, M.D., Frank Dunton, M.D., S. R. Lewis, M.D., and T. G. Blocker, Jr., M.D., Surgery Department, Division of Plastic Surgery, University of Texas Medical Branch, Galveston, Texas.

The history of this procedure was given. The technique of performing subcutaneous lymphangiography in the lower extremity was illustrated in considerable detail.

Material from about 50 examinations was used to demonstrate (1) normal lymphatics of the lower leg and thigh; (2) abnormal lymphatics associated with idiopathic lymphedema, varicose veins, chronic ulcers and a few miscellaneous conditions; and (3) normal inguinal and in some instances pelvic lymph nodes.

Other excellent exhibits were:

Intracranial Sarcomas: Their Roentgenographic Manifestations. Hillier L. Baker, Jr., M.D., Mayo Clinic, Rochester, Minnesota.

Primary intracranial sarcomas may be divided into five histologic groups: fibrosarcoma, perivascular sarcoma, reticulum cell sarcoma, hemangiopericytoma and a miscellaneous group. These neoplasms represent about 2 per cent of all primary intracranial tumors.

The roentgenographic alterations observed in a group of 54 such tumors were presented and two findings which may be somewhat specific for this type of neoplasm were emphasized.

Angiographic Demonstration of Extracranial Lesions Producing Cerebral Arterial Insufficiency. Curtis H. Burge, M.D., E. Stanley Crawford, M.D., and William S. Fields, M.D., Methodist Hospital and Baylor University College of Medicine, Houston, Texas.

The most common locations of obstructing lesions in the extracranial portions of arteries supplying the brain were shown diagrammatically and the lesions were illustrated by appropriate angiographic techniques. Brief clinical summaries were given and angiograms following operations or the relief of obstructions in a number of patients were included.

Percutaneous Femoral Catheterization of the Left Ventricle for Contrast Cardiovascular Visualization. Charles T. Dotter, M.D., University of Oregon Medical School, Portland, Oregon.

The scope of cardiovascular diagnosis by means of contrast visualization techniques has been greatly expanded. Percutaneous femoral guided catheterization of the left ventricle and systemic arteries is simple, safe and of proved worth in the direct demonstration of interventricular septal defects, mitral insufficiency and coronary heart disease. This technique was described and the results were illustrated by log-Etronic roentgenographic reproductions.

The two main advantages of the method are that it: (1) provides practical means for catheterization and selective visualization of the left ventricle, as well as of the aorta and many of its branches, through percutaneous femoral artery needle puncture; and (2) can be employed on an out-patient basis without requiring general anesthesia.

Anomalies of Intestinal Rotation and Fixation: Embryology and Radiological Features. R. L. Estrada, M.D., J. W. McKay, M.D., and D. J. Sieniewicz, M.D., The Montreal General Hospital, Montreal, Quebec, Canada.

A classification of anomalies of rotation and fixation of the mid-gut loop was given. The normal embryology was briefly outlined and the abnormalities were explained. Cases showing the typical features were presented and illustrated by diagrams, drawings, photographs of the gross specimens and pertinent roentgenograms.

Radiation Dose Reduction by Combined Copper and Aluminum Filters. Lewis E. Etter, M.D., Adolph G. Kammer, M.D., Herman Cember, M.S., and Lawrence C. Cross, R. T., School of Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania.

This exhibit was designed to show the combinations of filters, roentgenographic techniques, screens and films which produce the lowest possible dose of radiation to the patient consistent with completely diagnostic roentgenograms. It was demonstrated that this may be accomplished by using various thicknesses of copper and aluminum filters in combination, along with different qualities of films and kinds of intensifying screens. Measurements on a suitable mannikin revealed reduction of dose in conventional fluoroscopy of 88 per cent and of depth dose as to bone marrow up to 76 per cent.

Idiopathic Periureteric Fibrosis. John A. Evans, M.D., Thane Asch, M.D., John Pearce, M.D., and Peter Dineen, M.D., The New York Hospital—Cornell Medical Center, New York, New York.

In 1948 Ormond described the first patient with a disease characterized by the presence of a thin fibrous plaque which originated in the retroperitoneal space and which incorporated, according to its extent, the ureters and great vessels of the abdomen. Since that time, 35 cases have been reported in the literature.

This exhibit presented the clinical, pathologic and roentgenographic findings in 3 additional patients afflicted with idiopathic periureteric fibrosis which segmentally involved one or both ureters.

Familial Recurring Polyserositis. William R. Eyler, M.D., Robert J. Priest, M.D., and Robert K. Nixon, M.D., Henry Ford Hospital, Detroit, Michigan.

Familial recurring polyserositis is a disease almost entirely limited to persons of Armenian, Syrian or Jewish ethnic origin. The previous concept of this disorder as a

benign peritonitis should be broadened to include a polyserositis with recurring clinical manifestations resulting from inflammation of the peritoneum, pleura, meninges or joints. This disorder is not sex-linked and is apparently transmitted by a dominant gene of incomplete penetrance. Although this is a clean-cut syndrome, the pathogenesis is not known.

The patients have recurring episodes of severe abdominal, chest or joint pain with fever, leukocytosis and physical signs corresponding to the particular serosal membrane involved. The onset may be from early infancy to middle life.

The roentgen findings are nonspecific. During the acute attack, there is commonly a small intestinal ileus. Study of the chest during attacks of pleural pain demonstrates basilar atelectasis, sometimes accompanied by small or moderate amounts of pleural fluid and elevation of the diaphragm. Examinations between attacks of pain are within normal limits except for occasional enlargement of the spleen.

The disorder was observed in 20 patients.

Fractures and Dislocations of the Cervical Spine; Mechanisms of Injury. H. F. Forsyth, M.D., and Joseph E. Whitley, M.D., Department of Radiology, The Bowman Gray School of Medicine, Winston-Salem, North Carolina.

The mechanism of cervical spine injury is of greatest importance in the diagnosis and care of patients with cervical spine injuries. The more common mechanism injuries were illustrated schematically and in result were correlated with cases typifying each type. The incidence of each mechanism was analyzed in a series of 159 cases.

The following conclusion was rendered: The dynamic classification of cervical spine injuries provides the best method of correlation of the clinical, neurologic, roentgenographic and pathologic findings. A better understanding of the anatomic abnormalities will result in improved patient care.

Experimental Induction of Calcium Deposition in Human Rachitic Cartilage. Donald Fraser, M.D., Ph.D., John D. Munn, D.R.D., T. R., Joan M. Leeming, M. B., Ch.B., D.C.H., *et al.*, The Research Institute, The Hospital for Sick Children, Toronto, Ontario, Canada.

This exhibit comprised the results of the work done on experimental calcification of rachitic cartilage in the human. The following is the interpretation of the results obtained:

1. In two types of refractory rickets (rickets due to vitamin D deficiency and hypophosphatemic vitamin D refractory rickets), mineral deposition was induced in rachitic cartilage *in vitro* without vitamin D by establishing physiologic concentrations of Ca and P in surrounding medium.

2. Massive doses of vitamin D are not essential for calcification of rachitic cartilage in refractory rickets of simple type.

3. Vitamin D is not essential for mineral deposition in rachitic cartilage of infants with vitamin D deficiency.

4. Oral phosphate supplementation may be a useful adjunct in treatment of hypophosphatemic vitamin D refractory rickets. However, efficacy of prolonged therapy is not established.

5. The renal tubules in refractory rickets of simple type possess potential for efficient phosphate reabsorption—no intrinsic tubular dysfunction. The responses suggest that hyperphosphaturia and hypophosphatemia may be due to secondary hyperparathyroidism.

6. Lack of hypophosphaturic response is consistent with an intrinsic defect of P reabsorption by proximal tubules.

Intravenous Cholangiography. Abraham Geffen, M.D., and Martin H. Floch, M.D., Beth Israel Hospital, New York, New York.

The purpose of this exhibit was to summarize the results of 223 intravenous cholangiographies with a very detailed correlation of clinical, laboratory, surgical and roentgenographic findings. Surgery was performed on 102 patients with 89 per cent accuracy of diagnosis of common duct calculi.

Intravenous Aortography. Richard H.

Greenspan, M.D., Eugene F. Bernstein, M.D., and Merle K. Loken, M.D., University of Minnesota Hospitals, Minneapolis, Minnesota.

The thoraco-abdominal aorta and its major branches can be satisfactorily visualized by the rapid intravenous injection of large amounts of concentrated opaque agents. This obviates the need for direct, blind aortic puncture with its inherent dangers.

The technique was demonstrated, along with examples of thoracic and abdominal aortic aneurysms, occlusions of the aorta, the iliac and femoral arteries, renal arteriograms and nephrograms.

The radioactivity circulation time—arm vein to thoracic or abdominal aorta—was determined by preliminary injection of 50μc of I^{131} renografin (2 cc. stable renografin 76 per cent plus 60 cc. saline). The main injection consisted of 60–80 cc. of 90 per cent diatrizoate (hypaque or renografin).

The results in 115 abdominal aortographies and 30 thoracic aortographies were reviewed. No deaths or serious complications occurred. The method proved diagnostic in over 90 per cent of the cases.

Serial Femoral Arteriography in Occlusive Disease: Clinical-Roentgenologic Considerations. Henry Haimovici, M.D., Jerome H. Shapiro, M.D., and Harold G. Jacobson, M.D., Montefiore Hospital, New York, New York.

This exhibit evaluated the results in approximately 100 serial femoral arteriographies performed in patients with occlusive arterial disease. In the majority of the cases, a newly designed automatic long-segment serialograph was employed which has been in use since October, 1957. This unit is considered uniquely suitable for serial arteriography. Its operation was demonstrated and its advantages emphasized.

A working classification, encompassing the sites and extent of occlusive disease processes in the femoral-popliteal arterial system, was presented. In correlating the clinical, operative, and roentgenologic findings, special attention was paid to the amount and extent of the collateral circulation and the type and significance of the "run-offs." The two major groups studied were diabetic and nondiabetic arteriosclerosis. Distinguishing patterns in the two groups were observed and their clinical and surgical implications discussed.

Experience with Laminagraphy in a Small General Hospital—310 Patients. Leland E. Holly, II, M.D., Leland E. Holly, M.D., Arthur H. Joistad, M.D., and Everett H. Johnston, M.D., Hackley Hospital, Muskegon, Michigan.

This exhibit reviewed the technique and findings in 310 patients in whom laminagraphy was used. Axial-transverse laminagraphy was not employed.

The disease processes investigated or amenable to investigation were tabulated and the limitations of the method were pointed out. Conclusions were drawn as to indications and usefulness in a small hospital.

A detailed bibliography was furnished in mimeographed form.

The Sella Turcica. C. B. Holman, M.D., H. L. Baker, Jr., M.D., and A. H. Bulbulian, D.D.S., Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

A variety of intracranial lesions betray their presence by alterations in the sella turcica. These changes usually include one or more of the following processes: (1) enlargement of the sella; (2) destruction of surrounding bone; (3) abnormal calcifications about the sella; and (4) multiple alterations of the sella.

This exhibit illustrated by means of models, roentgenograms, and laminagrams each of these basic types of changes and demonstrated how, alone or in combination, the proper analysis of these features may suggest a satisfactory differential diagnosis and in certain instances indicate the proper additional roentgenographic procedures which might establish a definite diagnosis.

A teaching Exhibit on Congenital Heart Disease. Dixon L. Hughes, M.D., Leo G.

Rigler, M.D., and B. J. O'Loughlin, M.D., University of California, Los Angeles, California.

This exhibit presented 6 cases of various congenital heart lesions which have been diagnosed by roentgenographic methods and proved at surgery or autopsy. For each of the 6 cases (which were representative of the more commonly encountered lesions) plain roentgenograms, individual frames from angiocardiograms, and drawings of the defect found at surgery or autopsy were presented, and comments regarding anatomic-physiologic correlation were made. The 6 cases included: (1) primary pulmonary hypertension; (2) interventricular septal defect; (3) intra-atrial septal defect; (4) pulmonary valvular stenosis; (5) patent ductus arteriosus; and (6) tetralogy of Fallot.

The "Normal" Lumbosacral Spine. Major Francis Kruse, Jr., USAF (MC), Capt. Neil E. Crow, USAF (MC), and Capt. Byron C. Brogdon, USAF (MC), The United States Air Force Medical Service, Armed Forces Institute of Pathology, Washington, D. C.

This exhibit was based on a review of lumbosacral spine roentgenograms of 936 healthy, asymptomatic Air Force Academy and Air Cadet applicants, aged seventeen to twenty-seven years. Appropriate statistics, data and illustrative roentgenograms were provided to demonstrate the extreme frequency of apparent abnormalities in such a normal population. Interpretation of these roentgenograms had been made by the double reading technique.

Subcutaneous Ossification of the Legs: Report on 60 Cases Associated with Chronic Venous Insufficiency. Heinz I. Lippmann, M.D., and Ralph R. Goldin, M.D., Albert Einstein College of Medicine, Bronx, New York.

Subcutaneous ossification is a frequent late complication of chronic venous insufficiency of the legs. Sixty cases were reviewed. The soft tissue roentgenograms, venograms and laminagrams of 4 of these were presented, together with the photomicrographs from specimens removed surgically. The exhibit illustrated that the soft tissue ossification in this condition only involves the subcutaneous layer and is independent of the veins. A correlation with chronic venous insufficiency was made.

Roentgen and Hematological Manifestations of the Congenital Hemolytic Anemias. Joseph J. Litschgi, M.D., Cook County Hospital and Hektoen Institute of Medical Research, Chicago, Illinois.

This exhibit presented a teaching demonstration of the primary congenital hemolytic anemias to expedite early diagnosis and prompt treatment. The roentgen manifestations were stressed on the basis of hematologic confirmation. Condensed historical background and current classification were outlined. A summary was given of the clinical signs and symptoms, with a brief discussion of the pathogenesis and laboratory findings.

Examples were shown of: (1) sickle cell anemia; (2) Mediterranean anemia; (3) hereditary spherocytosis; and (4) sicle cell hemoglobin C disease with *Salmonella* osteomyelitis.

Case of the Day. William McAlister, M.D., Raymond Kurtzman, M.D., Jerome Wiot, M.D., Aaron Weinstein, M.D., and Alfred Lessure, M.D., Department of Radiology, Cincinnati General Hospital, Cincinnati, Ohio.

In this exhibit the history, clinical data and roentgenograms of an unusual case were presented as the "case of the day."

The participating contestants would make a diagnosis and place this in a sealed box. The following morning the list of the "winners" was posted.

Myelofibrosis. William Meszaros, M.D., and Melvin Sisson, M.D., Cook County Hospital, Chicago, Illinois.

Five cases were presented and the criteria for the roentgen diagnosis of myelofibrosis were discussed. Radiolucent lesions, as well as osteosclerosis, are an important feature. The close relationship to myeloid leukemia was emphasized.

Respiratory Distress in the Newborn. Vic-

tor G. Mikity, M.D., and George Jacobson, M.D., University of Southern California School of Medicine and Los Angeles County Hospital, Los Angeles, California.

Respiratory distress in the newborn can be attributed to many causes. Only those of pulmonary origin were considered in this exhibit: hyaline membrane syndrome, aspiration of amniotic fluid, *Staphylococcus* pneumonia, interstitial mononuclear pneumonitis, necrotizing bronchopneumonia, tuberculosis, adenomatoid malformation, pulmonary edema, lobar and interstitial emphysema. The roentgen manifestations of most of these entities can be strikingly similar.

It was stressed that the above mentioned conditions cannot be differentiated on roentgenologic grounds alone, but that careful consideration must be given to the history, physical and laboratory findings.

Intra-Aortic Nitrogen Mustard Used in Palliation of Advanced Pelvic Malignancy. G. Melvin Stevens, M.D., Sydney F. Thomas, M.D., and Blake C. Wilbur, M.D., Palo Alto Medical Clinic, Palo Alto, California.

The intra-aortic administration of nitrogen mustard for the relief of intractable symptoms of advanced pelvic malignancy was described and illustrated. The following is an outline of the procedure: (1) the patient is premedicated; (2) the procedure is done on the roentgenographic table; (3) percutaneous femoral artery puncture is performed using a No. 18 gauge needle; (4) a No. 60 polyethylene catheter is threaded to the level of the second lumbar interspace and the position checked; (5) 20 mg. of HN_2 is injected; and (6) pneumatic tourniquets occlude the femoral artery flow for a three minute period.

This method can be used alone or in combination with radiation therapy and may be repeated with safety. It appears to have a particular application in radioresistant tumors.

Approximately 50 patients have been treated with 83 injections. Only a few minor complications were observed: femoral arteriospasm in 1 patient; transient hypertension in 1 patient; temporary elevation of temperature in 3 patients; and curling of the catheter in the femoral artery in 3 patients.

The effect is generally rapid and the results are encouraging.

Postbulbar Duodenal Obstruction. William C. Strittmatter, M.D., and Charles H. Brown, M.D., Cleveland Clinic and Cleveland Clinic Hospital, Cleveland, Ohio.

In this exhibit multiple causes for obstruction distal to the duodenal bulb were demonstrated. A dilated duodenal bulb may be the only roentgen sign. The cause of obstruction may not be apparent from the roentgenologic examination. Surgery may be necessary. In benign lesions duodenojejunostomy is the operative method of choice.

Iridium 192 in Cancer Therapy. Herman D. Suit, M.D., Stanley E. Sneider, M.D., Robert W. Swain, B. S., and J. Robert Andrews, M.D., Radiation Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland.

This exhibit pertained to the use of iridium 192 in the treatment of human cancer. Special instruments have been developed for holding and placing iridium 192 sources as both permanent and removable implants. Techniques were demonstrated for diminution of the radiation exposure of the operator to well below maximum permissible exposure limits. Comparative data were given for operator exposure during radium needle, cobalt 60 wire and iridium 192 wire implantation.

Roentgen Manifestations of Mesenteric Vascular Disease. C. C. Wang, M.D., and J. D. Reeves, M.D., Massachusetts General Hospital, Boston, Massachusetts.

Mesenteric vascular disease is not a clear-cut roentgenographic entity and may present in a variety of patterns. Cases of malabsorption syndrome, regional enteritis, ulcerative colitis and localized tumefaction may have their origin in vascular disease. Because the bowel lends itself so admirably to roentgenologic study, these possibilities

should be kept in mind. In addition to plain roentgenography and barium studies, aortography and mesenteric arteriography may aid in establishing the diagnosis and further may be of valuable assistance in clinical management.

Roentgen Findings in Histoplasmin-Positive School Children. Walter M. Whitehouse, M.D., John F. Holt, M.D., Winthrop N. Davey, M.D., and Otto K. Engelke, M.D., University Hospital, Ann Arbor, Michigan.

Discovery of an endemic area of histoplasmosis within 20 miles of the University Hospital has offered the opportunity of studying the various roentgen manifestations of the active and inactive primary infection in histoplasmin-positive tuberculin-negative school children in a school population with nearly 90 per cent incidence of positive skin tests by the time the children have reached the age of fourteen. A selected group with active primary complexes has been followed for one year to observe the natural course of the disease.

The tabulated data obtained from examining over 1,000 children were presented with illustrated case material. Negative chest roentgenograms were found in 75 per cent of histoplasmin-positive and tuberculin-negative school children. Two-thirds of school children with positive roentgen findings showed that the process already progressed to a stage of parenchymal or nodal calcification or both. Unilateral hilar calcification was the most frequent abnormality seen, with a slightly lower incidence of solitary parenchymal calcification or a combination of these findings. Multiple parenchymal calcifications occurred less commonly than the solitary pulmonary calcification. Of the earlier uncalcified lesions, hilar lymphadenopathy, nodular lesions and infiltrative lesions were found in approximately equal proportion. Combinations of calcified and uncalcified lesions may occur. Pleural involvement is relatively minor and is infrequently seen. Of 52 cases with uncalcified primary lesions, 37 showed evidence of regression or calcification on follow-up, 13 showed no change, and only 2 showed evidence of progression of the disease.

It seems probable that further intensive skin testing will uncover additional localized endemic areas which are not now recognized. Further study of these areas will add to our knowledge of the epidemiology of histoplasmosis.

Colonic Endometriosis. Fred K. Wietersen, M.D., Ross M. Balow, M.D., Leo S. Figiel, M.D., Steven J. Figiel, M.D., and Desmond K. Rush, M.D., Grace Hospital, Detroit, Michigan.

Types of deformities of the colon produced by endometriosis were illustrated. The two most frequently seen are pseudopolypoid defects and stenosing lesions in the rectosigmoid. Typically, the mucosa is intact. At times, complete obstruction may be produced. This disease process is becoming more common in the female and it behooves the radiologist to bear it in mind. The diagnosis will be made more readily by taking into consideration not only the roentgenologic manifestations but the clinical history and physical findings as well.

The Esophagogastric Closing Mechanism (The Role of the Abdominal Portion of the Esophagus). Bernard S. Wolf, M.D., The Mount Sinai Hospital, New York, New York.

The intrinsic sphincteric action of the terminal portion of the esophagus is not sufficient to maintain esophagogastric closure with the patient in the inverted or Trendelenburg position. An extrinsic factor at the esophageal hiatus of the diaphragm is required. An analysis of the hydrostatic features indicates that the "diaphragmatic factor" is the result of the action of the hydrostatic forces on a submerged segment, that is, the abdominal portion of the esophagus.

The existence of such a portion of the esophagus was proved by opaque markers, roentgen and intraluminal pressure studies. A contracted herniated portion of the stomach may act in a similar but less efficient fashion.

THE COMMERCIAL EXHIBITS

THE Commercial Exhibits of the Sixtieth Annual Meeting of the American Roentgen Ray Society, held at the Netherland Hilton Hotel, Cincinnati, Ohio, September 22–25, 1959, undoubtedly were the best in the Society's history. The rapid technical evolution of the last few years has brought forth amazing advances in the design and construction of equipment for uses in all branches of radiology. This was amply evident in the many individual exhibits which, by their most artistic arrangement, greatly enhanced the concept of "progress —beautifully displayed." They evoked enthusiastic admiration from the large number of visitors, which must have been greatly rewarding to the exhibitors. The unusual facilities of the Netherland Hilton Hotel and the tireless work of Mr. Clifford L. Sherratt, Honorary Member, and Dr. James C. Cook, Manager of the Annual Meeting, were additional factors which have contributed to making this year's Commercial Exhibits so attractive and eminently successful. The Society extends to all exhibiting firms its profound appreciation and sincerest thanks.

The following is the list of the firms which participated in the Commercial Exhibits:

Abbott Laboratories, North Chicago, Illinois; Allis-Chalmers Manufacturing Company, Milwaukee, Wisconsin; Ansco, Binghamton, New York; Automatic Seriograph Corporation, College Park, Maryland; Barnes-Hind Barium Products Company, Sunnyvale, California; Carr Corporation, Culver City, California; Dick X-Ray Company, St. Louis, Missouri; Dietene Company, Minneapolis, Minnesota; E. I. du Pont de Nemours & Company, Inc., Wilmington, Delaware; Eastman Kodak Company, Rochester, New York; Eureka X-Ray Tube Corporation, Chicago, Illinois; Fairchild Camera and Instrument Corporation, Long Island, New York; Oscar Fisher Company, Inc., Newburgh, New York; E. Fougera & Company, Inc., Long Island, New York; Franklin X-Ray Corporation, Philadelphia, Pennsylvania; General Electric Company, Milwaukee, Wisconsin; M. J. Gordon Consultants, New York, New York; Gray Pharmaceutical Company, Inc., New York, New York; Great Books of the Western World, Grand Rapids, Michigan; Grune & Stratton, Inc., New York, New York; Halsey X-Ray Products, Inc., Brooklyn, New York; Paul B. Hoeber, Inc., New York, New York; Hospital Microfilming, Pearl River, New York; Howdon Videx Products Corporation, Mount Vernon, New York; Philip A. Hunt Company, Palisades Park, New Jersey; Ilford Inc., New York, New York; Keleket X-Ray Corporation, Waltham, Massachusetts; Leishman X-Ray Engineering Company, Los Angeles, California; Liebel-Flarsheim Company, Cincinnati, Ohio; LogEtronics Inc., Alexandria, Virginia; Low X-Ray Film Corporation, New York, New York; Machlett Laboratories, Inc., Springdale, Connecticut; Mallinckrodt Chemical Works, St. Louis, Missouri; Mattern X-Ray Division of Land-Air, Inc., Chicago, Illinois; Micro X-Ray Recorder, Inc., Chicago, Illinois; North American Philips Company, Inc., New York, New York; Nuclear-Chicago Corporation, Chicago, Illinois; Pako Corporation, Minneapolis, Minnesota; Pelvic Anchor Corporation, Rochester, New York; Physicians' Technical Equipment Company, Milwaukee, Wisconsin; Picker X-Ray Corporation, White Plains, New York; Profexray, Inc., Maywood, Illinois; Schick X-Ray Company, Inc., Chicago, Illinois; Frank Scholz X-Ray Corporation, Boston, Massachusetts; Siemens New York, Inc., New York, New York; E. R. Squibb & Sons, New York, New York; Standard X-Ray Company, Chicago, Illinois; Charles C Thomas, Publisher, Springfield, Illinois; Thuresson-Angrabright, Skokie, Illinois; United States Radium Corporation, Morristown, New Jersey; Westinghouse Electric Corporation, Pittsburgh, Pennsylvania; Winthrop Laboratories, New York, New York; X-Ray Solution Service, Detroit, Michigan; Year Book Publishers, Inc., Chicago, Illinois.

SOCIETY PROCEEDINGS AND NEWS ITEMS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. C. Allen Good, Mayo Clinic, Rochester, Minn. Annual meeting: Ambassador Hotel, Atlantic City, N. J., Sept. 27–30, 1960.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Robert L. Brown, Winship Clinic, Emory University, Atlanta 22, Ga. Annual meeting: Caribe Hilton, San Juan, Puerto Rico, March 17–19, 1960.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 713 E. Genesee St., Syracuse 2, N. Y. Annual meeting: Palmer House, Chicago, Ill., Nov. 15–20, 1959.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Illinois. Annual meeting: Feb. 3–6, 1960, Roosevelt Hotel, New Orleans, La.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. T. Leucutia, Harper Hospital, Detroit 1, Mich. Annual meeting: June 13–17, 1960, Miami Beach, Fla.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building. Rochester, Minn. The Board will conduct a Special Examination in Nuclear Medicine (for diplomates in Radiology or Therapeutic Radiology) on Dec. 5, 1959 at the Shoreham Hotel in Washington, D. C. Deadline for filing applications was Aug. 1, 1959. The regular fall examination will be held at the Shoreham Hotel, December 6–9, 1959. Deadline for filing applications was July 1, 1959. Candidates examined in Diagnostic Roentgenology may expect to be examined in Physics. The spring 1960 examination will be held at the Terrace Hilton Hotel, Cincinnati, Ohio, June 6–10; deadline for filing applications is January 1, 1960. A Special Examination in Nuclear Medicine (for diplomates in Radiology or Therapeutic Radiology) will be offered provided there are sufficient applications.

TENTH INTERNATIONAL CONGRESS OF RADIOLOGY
Secretary-General, Dr. Carleton B. Peirce, Royal Victoria Hospital, Montreal 2, Quebec, Canada. Meets in Montreal, Aug. 26–Sept. 1, 1962.

SEVENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Secretary, Dr. Laura Fariñas, Havana, Cuba. *Counselor for the United States*, Dr. J. A. del Regato, Penrose Cancer Hospital, 2200 North Cascade Avenue, Colorado Springs, Colorado. The meeting will be held in Brazil in 1961.

ALABAMA RADIOLOGICAL SOCIETY
Secretary, Dr. J. A. Meadows, Jr., Medical Arts Bldg., Birmingham 5, Ala. Meets time and place Alabama State Medical Association.

AMERICAN NUCLEAR SOCIETY
Executive-Secretary, Octave J. Du Temple, 86 E. Randolph St., Chicago, Ill.

ARIZONA RADIOLOGICAL SOCIETY
Secretary, Dr. R. Lee Foster, 1313 N. Second St., Phoenix, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. J. B. Scruggs, Arkansas Baptist Hospital, Little Rock, Ark. Meets every three months and also at time and place of State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary, Dr. Paul Riemenschneider, Medical College State University of New York, Syracuse, N. Y. Annual meeting to be announced.

ATLANTA RADIOLOGICAL SOCIETY
Secretary, Dr. J. L. Clements, Jr., 35 Linden Ave., N.E., Atlanta 8, Ga. Meets monthly, except during three summer months, on second Friday evening.

BLOCKLY RADIOLOGICAL SOCIETY
Secretary, Dr. Samuel Finkelman, 101 S. Twentieth St. Philadelphia, Pa.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary, Dr. George A. Manfredonia, One Hanson Place, Brooklyn, N. Y. Meets first Thursday of each month October through May.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Alfred Dobrak, 710 Leydecker Rd., Buffalo 24, N. Y. Meets second Monday evening each month, October to May inclusive.

CENTRAL NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph A. Head, 150 Marshall St., Syracuse, N. Y. Meets first Monday each month October through May.

CENTRAL OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. William B. Schwartz, 1500 W. Third Ave., Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, November, January, March and May at Fort Hayes Hotel, Columbus, Ohio.

CENTRAL SOCIETY OF NUCLEAR MEDICINE
Secretary, Dr. Robert S. Landauer, Radiation Center Building, 1903 West Harrison St., Chicago 12, Ill.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. George B. Cahill, 802 Burns Ave., Flossmoor, Ill. Meets second Thursday of each month, October to April except December at the Sheraton Hotel at 8:00 P.M.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. Frederick A. Rose, 2065 Adelbert Rd., Cleveland 6, Ohio. Meetings at 7:00 P.M. on fourth Monday of each month from October to April at Tudor Arms Hotel.

COLORADO RADIOLOGICAL SOCIETY
Secretary, Dr. Lorenz R. Wurtzebach, 601 E. Nineteenth Ave., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
Secretary, Dr. Paul J. Kingston, 114 Woodland St., Hartford, Conn. Meets first Friday in February and April.

DALLAS-FORT WORTH RADIOLOGICAL CLUB
Secretary, Dr. A. H. Keene, 3707 Gaston Ave., Dallas, Texas. Meets monthly, third Monday, at Greater Fort Worth International Airport at 6:30 P.M.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. Kenneth L. Krabbenhoft, Harper Hospital, Detroit 1, Mich. Meets monthly first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

EAST BAY ROENTGEN SOCIETY
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

EAST TENNESSEE RADIOLOGICAL SOCIETY
Secretary, Dr. Charles W. Reavis, Baroness Erlanger Hospital, Chattanooga, Tenn. Meets in January and September.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Eastern Radiological Society
Secretary, Dr. John D. Osmond, Jr., Euclid-Glenville Hospital, Cleveland 19, Ohio. Meets at Mid Pines Club, Southern Pines, N. C., March 6–10, 1960.

Florida Radiological Society
Secretary, Dr. Russell D. D. Hoover, 49th St., West Palm Beach, Fla. Meets twice annually, in the spring with the annual State Society Meeting, and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Joseph C. Rush, 1501 Jungle, St. Petersburg, Fla.

Georgia Radiological Society
Secretary, Dr. George W. Brown, Griffin, Ga. Meets in spring and fall with Annual State Society Meeting.

Greater Miami Radiological Society
Secretary, Dr. George P. Daurelle, Jackson Memorial Hospital, Miami 36, Fla. Meets monthly third Wednesday at 8 P.M. at Jackson Memorial Hospital, Miami, Fla.

Greater St. Louis Radiological Society
Secretary, Dr. Harvey A. Humphrey, 462 N. Taylor, St. Louis 8, Mo.

Houston Radiological Society
Secretary, Dr. Edward B. Singleton, 6621 Fannin St., Houston 25, Texas. Meets last Monday each month, Seminar Room, Doctors' Club of Houston.

Idaho State Radiological Society
Secretary, Dr. Charles R. McWilliams, Magic Valley Memorial Hospital, Twin Falls, Idaho. Meets annually at same time as Idaho State Medical Association.

Illinois Radiological Society
Secretary, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in spring and fall.

Indiana Roentgen Society
Secretary, Dr. David E. Wheeler, 1500 North Ritter, Indianapolis, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

Iowa Radiological Society
Secretary, Dr. James T. McMillan, 1104 Bankers Trust Bldg., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society
Secretary, Dr. Lewis G. Allen, 807 Huron Bldg., Kansas City, Kansas. Meets in spring with State Medical Society, and in winter on call.

Kentucky Radiological Society
Secretary, Dr. Robert H. Akers, V. A. Hospital, Louisville 2, Ky. Meets monthly on second Friday at Seelbach Hotel, Louisville.

Kings County Radiological Society
Secretary, Dr. C. P. Naidorf, 411 Parkside Ave. Brooklyn 26, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. Denis C. Adler, 2010 Wilshire Blvd., Los Angeles 57, Calif. Meets second Wednesday of month in September, November, March, April and June at Los Angeles County Medical Association Building, Los Angeles

Maine Radiological Society
Secretary, Dr. Edward C. Porter, 489 State Street, Bangor, Maine. Meets in June, September, December and April.

Maryland Radiological Society
Secretary, Dr. Nathan B. Hyman, 1805 Eutaw Place, Baltimore 17, Md.

Memphis Roentgen Society
Secretary, Dr. Hollis H. Halford, Kennedy V.A. Hospital, Department of Radiology, Memphis 15, Tenn. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. S. F. Johnson, 2197 Los Arrow Dr., Dayton 9, Ohio. Meets second Friday of fall and winter months.

Mid-Hudson Radiological Society
Secretary, Dr. Joseph Sorrentino, St. Francis Hospital, Poughkeepsie, N. Y. Meets 8:30 P.M., fourth Wednesday each month, September to May.

Milwaukee Roentgen Ray Society
Secretary, Dr. Joseph F. Wepfer, 5000 W. Chambers St., Milwaukee 10, Wis. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society
Secretary, Dr. Donald H. Peterson, 853 Medical Arts Bldg., Minneapolis 2, Minn. Meets three times annually, in fall, winter and spring.

Mississippi Radiological Society
Secretary, Dr. Bernard T. Hickman, University Medical Center, Jackson, Miss. Meets third Thursday of each month at Hotel Edwards, Jackson, at 6:00 P.M.

Montana Radiological Society
Secretary, Dr. Arthur T. Austin, 104 The Doctors Bldg., Billings, Montana. Meets at least once a year.

Nassau Radiological Society
Secretary, Dr. Jerome Zwanger, 126 Hicksville Rd., Massapequa, L. I., N. Y. Meets second Tuesday of the month in February, April, June, October and December.

Nebraska Radiological Society
Secretary, Dr. Warren Q. Bradley, 924 Sharp Bldg., Lincoln, Nebraska. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. John E. Gary, 1180 Beacon St., Brookline 46, Mass. Meets third Friday of each month, October through May, at The Longwood Towers, Brookline, Mass.

New Hampshire Roentgen Ray Society
Secretary, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. William B. Seaman, 622 W. 168th St., New York, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 4:30 P.M.

North Carolina Radiological Society
Secretary, Dr. Charles A. Bream, 211 McCauley St., Chapel Hill, N. C. Meets in May and October.

North Dakota Radiological Society
Secretary, Dr. Marianne Wallis, St. Joseph's Hospital, Minot, N. D. Meetings by announcement.

North Florida Radiological Society
Secretary, Dr. Paul A. Mori, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Irving Van Woert, Jr., Albany Hospital, Albany, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

Northern California Radiological Society
Secretary, Dr. Rob H. Kirkpatrick, 1219 28th St., Sacramento, Calif. Meets at dinner last Monday of each month, September to June.

Ohio State Radiological Society
Secretary, Dr. Paul D. Meyer, 125 S. Grant Ave., Columbus, Ohio. Annual meeting to be announced.

Oklahoma State Radiological Society
Secretary, Dr. J. Murphree, 617 E. Brookfield, Ponca City, Okla. Three regular meetings annually.

Oregon Radiological Society
Secretary, Dr. Barbara J. Radmore, 1162 Willamette, Eugene, Ore. Meets monthly from October to June on the second Wednesday of each month at 8:00 P.M. at the University Club.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. J. Arch Colbrunn, 19 Royal Oak Drive, Roseburg, Ore. Annual meeting: Vancouver, British Columbia, May, 1960.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco 8, Calif. Meets annually during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. Walter P. Bitner, 234 State St., Harrisburg, Pa.

Philadelphia Roentgen Ray Society
Secretary, Dr. Robert B. Funch, Department of Radiology, Germantown Hospital, Philadelphia 44, Pa. Meets first Thursday of each month, at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Charles N. Chasler, 3500 Fifth Ave., Pittsburgh 13, Pa. Meets second Wednesday of month, October through June at Park Schenley Restaurant.

Radiological Section, Baltimore Medical Society
Secretary, Dr. James K. V. Willson, 1100 N. Charles St., Baltimore 1, Md. Meets third Tuesday each month, September to May, inclusive.

Radiological Society of Greater Cincinnati
Secretary, Dr. John E. Singer, 468 Doctors' Bldg., Cincinnati 2, Ohio. Meets monthly from September to May on first Monday of each month at 7:30 P.M. at the Cincinnati General Hospital.

Radiological Society of Hawaii
Secretary, Dr. Louis L. Buzaid, The Queen's Hospital, Box 861, Honolulu, Hawaii. Meets third Monday of each month at 7:30 P.M.

Radiological Society of Greater Kansas City
Secretary, Dr. Samuel B. Chapman, Jr., 830 Argyle Bldg., Kansas City, Mo. Meets last Friday of each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. Seymour Ochsner, Ochsner Clinic, New Orleans 15, La. Meets annually during Louisiana State Medical Society meeting.

Radiological Society of New Jersey
Secretary, Dr. Austin J. Tidaback, 912 Prospect Ave., Plainfield, N. J. Meets at Atlantic City at time of State Medical Society meeting and in November in Newark, N. J.

Radiological Society of New York State
Secretary-Treasurer, Dr Mario C. Gian, 610 Niagara St.. Buffalo 1, N. Y. Annual meeting to be announced.

Radiological Society of South Dakota
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
Secretary, Dr. Joseph F. Linsman, 436 N. Roxbury Dr., Beverly Hills, Calif.

Redwood Empire Radiological Society
Secretary, Dr. Lee E. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Theodore F. Van Zandt, 501 W. Main St., Rochester 8, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. John H. Freed, 4200 East Ninth Ave., Denver 20, Colo. Annual meeting: Denver Hilton Hotel, Denver, Colo., Aug. 11–13, 1960.

San Antonio-Military Radiological Society
Secretary, Dr. Hugo F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Texas. Meets third Wednesday each month in Fort Sam Houston Officer's Club at 6:30 P.M.

San Diego Radiological Society
Secretary, Dr. Stanley A. Moore, 2466 First Ave., San Diego 1, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. M. A. Sisson, 450 Sutter St., San Francisco 8, Calif. Meets quarterly at Grison's Steak House.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Connecticut State Medical Society
Secretary, Dr. Ralph J. Littwin, Bristol Hospital, Bristol, Conn. Meetings are held bi-monthly.

Section on Radiology, Medical Society of the District of Columbia
Secretary, Dr. William E. Sheely, 1835 Eye St., N.W., Washington 6, D. C. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. William Meszaros, 1825 W. Harrison St., Chicago, Ill.

Section on Radiology, Southern Medical Association.
Secretary, Dr. Seymour Ochsner, Ochsner Clinic, 3503 Prytania St., New Orleans 15, La. Annual meeting: Nov. 16–19, 1959, Atlanta, Ga.

Shreveport Radiological Club
Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M. September to May inclusive.

Society of Nuclear Medicine
Secretary, Dr. Robert W. Lackey, 452 Metropolitan Bldg., Denver 2, Colo. Annual meeting to be announced.

South Bay Radiological Society
Secretary, Dr. Stanford B. Rossiter, 1111 University Dr., Menlo Park, Calif. Meets second Wednesday of each month.

South Carolina Radiological Society
Secretary, Dr. Wayne Reeser, 804 Burroughs St., Conway, S. C. One meeting with South Carolina Medical Association in May, others arranged by President.

Southern Radiological Conference
Secretary, Dr. Marshall Eskridge, Mobile Infirmary, Mobile, Ala.

Southwestern Radiological Society
Secretary, Dr. Gordon Black, Suite 2-A, El Paso Medical Center, 1501 Arizona, El Paso, Texas. Meets second Tuesday of each month.

Tennessee Radiological Society
Secretary, Dr. James J. Range, P.O. Box 324, Johnson City, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 402 Professional Bldg., 1216 Pennsylvania Ave., Fort Worth 4, Texas. Next meeting January 22 and 23, 1960, Houston, Texas.

Tri-State Radiological Society
Secretary, Dr. James R. Mathews, 118 S. E. First St., Evansville, Ind. Meets last Wednesday of Oct., Jan., March and May, 8:00 P.M. at Elks' Club in Evansville.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society
Secretary, Dr. William R. Christensen, 2033 S. State St., Salt Lake City, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Virginia Radiological Society
Secretary, Dr. Frank A. Kearney, II, 110 S. Curry St., Phoebus, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Wayne A. Chesledon, 306 Stimson Bldg., Seattle 1, Wash. Meets fourth Monday of each month from September through May at 610 Pine St., Seattle.

West Virginia Radiological Society
Secretary, Dr. Karl J. Myers, 112 N. Woods St., Philippi, W. Va. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester Radiological Society
Secretary, Dr. James C. Montieth, 170 Maple Avenue, White Plains, N. Y. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society
Secretary, Dr. Howard G. Bayley, 116 Iroquois Parkway, Beaver Dam, Wis. Annual Meeting each spring in various places.

X-Ray Study Club of San Francisco
Secretary, Dr. John H. Heald, 450 Sutter St., San Francisco 8, Calif. Meets monthly, third Thursday at 7:30 P.M., Children's Hospital, September through June.

Cuba, Mexico, Puerto Rico and Central America

Asociación de Radiólogos de Centro America y Panamá
Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá, *Secretary-General*, Dr. Roberto Calderón, Calle Central Deste No. 218, Managua, Nicaragua. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología de El Salvador
Secretary, Dr. Rafael Vega Gómez.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad de Radiología y Fisioterapía Cubana
Secretary, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

Sociedad Costarricense de Radiologia
Secretary, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.

Sociedad Mexicana de Radiología, A. C.
Calle del Oro No. 15, México 7, D. F.
Secretary-General, Dr. Guillermo Santín. Meets first Monday of each month.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 86, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital, chosen at preceding meeting.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Secretary, Dr. Odilon Raymond, 5400 Blvd. Gouin. Quest, Montreal, Que. Meets four times a year.

British Institute of Radiology Incorporated with the Röntgen Society
Honorary Secretary, Dr. John Blewett, 32 Welbeck St., London, W. 1. Meets monthly from October until May.

Faculty of Radiologists
Honorary Secretary, Dr. R. A. Kemp Harper, 47 Lincoln's Inn Fields, London, W.C.2, England.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1.

Canadian Association of Radiologists
Honorary Secretary, Dr. Robert G. Fraser, *Associate Honorary Secretary*, Dr. Jean-Louis Léger, 1555 Summerhill Ave., Montreal 25, Que. Meetings January and June.

Montreal Radiological Study Club
Secretary, Douglas W. MacEwan, 2300 Tupper St., Montreal 25, Quebec. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electro-Radiologie Médicale
General Secretary, Dr. Louis Ivan Vallée, 1058 rue St-Denis, Montreal 18, Canada. Meets third Saturday each month.

Toronto Radiological Society
Secretary, Dr. L. R. Harnick, Toronto Western Hospital, 399 Bathurst St., Toronto, Ontario. Meets second Monday of each month September through May.

College of Radiologists of Australasia
Honorary Secretary, Dr. E. A. Booth, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Victor A. Añaños, Instituto de Radiologia, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional del Centenario, Santa Fe 1300, Rosario.

O Colégio Brasileiro de Radiologia
Secretary-General, Dr. Walter Bomfins Pontes. Seat: Rua Major Quedinho, 99-2°. Andar, São Paulo, Brazil.

Sociedad Argentina de Radiología, Junta Central, Buenos Aires
Secretary, Dr. Edgardo O. Olcese, Santa Fé 1171, Buenos Aires. Meetings are held monthly.

Sociedad Bolivana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1596, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Juan Sabbagh Dacla, Merced 565, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary, Dr. Alberto Mejía Diazgranados, Carrera 13, No. 25–31, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología y Fisioterapía
Secretary, Dr. Publio Vargas P., Casilla 1242, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary, Dr. Luis Pinillos Ganoza, Apartado 2306, Lima, Perú. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 218, Lima.

Sociedad de Radiologia del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Ernesto H. Cibils, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiologia de Pernambuco
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Rubén Merenfeld, Apartado No. 9362, Candelaria, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 256 Chaussée de Wavre, Heverlee-lez-Louvain, Belgium. Meets in February, March, May, June, October, November and December.

Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris.
Secretary-General, Dr. Ch. Proux, 9, rue Daru, Paris 8e, France.

Československá Společnost pro Roentgenologii a Radiologii
Secretary, Dr. Robert Poch, Praha 12, Šrobárova 50, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Deutsche Röntgengesellschaft
Secretary, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut. Lagenbeckstr. 1, Mainz, Germany. Meets annually.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Electrologie en Röntgenologie
Secretary, Dr. J. R. von Ronnen, Violenweg 14, den Haag, Netherlands.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Española de Radiologia y Electrologia
Secretary, Dr. D. Aureo Gutierrez Churruca, Esparteros, No. 9, Madrid, Spain. Meets monthly in Madrid.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

India

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

The next list of Meetings of Radiological Societies will be published in the January issue of the Journal.

AMERICAN ROENTGEN RAY SOCIETY ELECTS NEW OFFICERS

At the meeting held at The Netherland Hilton, Cincinnati, Ohio, on September 22–25, 1959, the following officers were elected: President, E. B. D. Neuhauser, Boston, Mass.; President-Elect, Harold G. Reineke, Cincinnati, Ohio; 1st Vice-President, Fred O. Coe, Washington, D. C.; 2nd Vice-President, Frederic E. Templeton, Seattle, Wash.; Secretary, C. Allen Good, Rochester, Minn.; and Treasurer, Robert K. Arbuckle, Oakland, Calif.

The next meeting will be held September 27–30, 1960, at the Ambassador Hotel, Atlantic City, New Jersey.

THE ROCKY MOUNTAIN RADIOLOGICAL SOCIETY

At a recent meeting of the Rocky Mountain Radiological Society, the following new officers for 1960 were elected: President-Elect, Peter E. Russo; 1st Vice-President, John W. Walker; 2nd Vice-President, Maurice C. Archer; Secretary-Treasurer, John H. Freed; Executive Committee, Mark S. Donovan; Program Committee, Ralph C. Moore.

The next Annual Meeting will be held in Denver, Colorado, on August 11, 12, and 13, 1960 at the Denver Hilton Hotel.

THE ROBERT J. REEVES LECTURE

On October 16, 1959, Dr. Charles L. Martin of Dallas, Texas, gave the third Robert J. Reeves Lecture at Duke Medical Center, Durham, North Carolina. Dr. Martin spoke on the treatment of cancer of the head and neck with irradiation.

JAMES PICKER FOUNDATION ADDS NEW FELLOWSHIP TO ITS PROGRAM

The new *James Picker Foundation Advanced Fellowships in Academic Radiology* now are offered in addition to the previously established research fellowships and scholar and research grants, which will be continued during 1960–1961. The new awards are planned to prepare the candidate to meet in full the intellectual demands of an academic position in radiology. Emphasis is placed upon the acquisition of a broad background in the basic sciences related to radiology. In general, it is expected that the candidate will be not more than thirty-three years of age and will have completed his clinical training. He should be prepared to devote a minimum of two years to course work in the basic sciences, to the application of the techniques and methods of these basic disciplines to radiological research, and to other activities designed to enhance his preparation for an academic career. The details of his program will be worked out on an individual basis through consultation between the candidate, his clinical and academic advisers, and the members of the Academy–Research Council's Committee on Radiology. Applications will be accepted on nomination by a clinical adviser, who should be a member of the staff of a department of radiology in a medical school.

The previously established program of the Foundation, for which applications again are being invited, comprises three types of awards: (1) *Postdoctoral Fellowships in Radiological Research;* (2) *Grants for Scholars;* and (3) *Grants-in-Aid of Research.*

Applications in these four categories will be reviewed by the Committee on Radiology of the Academy–Research Council's Division of Medical Sciences. Final determination of awards is made by the Foundation upon recommendation of the Division. Support is not restricted to citizens of the United States or to laboratories within this country.

Applications for the fiscal year 1960–1961 should be submitted by December 15, 1959. Further details and application blanks may be obtained from the *Division of Medical Sciences—Room 411, National Academy of Sciences–National Research Council, 2101 Constitution Avenue, N.W., Washington 25, D. C.*

The National Research Council of Canada serves as scientific adviser to the James Picker Foundation with respect to its Canadian program. Accordingly, if the proposed studies are to be carried out in Canada, requests for information and application forms should be directed to the Awards Office, National Research Council of Canada, Ottawa 2, Canada.

FIRST KIRKLIN-WEBER MEMORIAL LECTURE

The First Kirklin-Weber Memorial Lecture will be held in Rochester, Minnesota, at 8:15 P.M., Friday, November 20, 1959. The lecturer on this occasion will be Dr. Leo G. Rigler, former Professor of Radiology of the University of Minnesota, and at present Executive Director of Cedars of Lebanon Hospital of Los Angeles, California. Doctor Rigler will speak on the subject "History of Roentgenology of the Gastrointestinal Tract."

The Kirklin-Weber Memorial Fund, which supports this lectureship, was contributed by former students and friends of the late B. R. Kirklin and H. M. Weber.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Bronchography. By C. Dijkstra, M.D., Medical Superintendent of "De Klokkenberg," Sanatorium and Chest Hospital, Breda, The Netherlands. Cloth. Price, $9.50. Pp. 157, with 106 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1958.

This brief and concise little monograph deals solely with the technique and interpretation of results obtained in bronchography. The text is essentially a group of well selected illustrative case reports, each accompanied by large, clear roentgenographic reproductions. The cases are grouped to include bronchial changes due to foreign bodies, malignant tumors, granulomas, asthma, bronchitis, and bronchiectasis. In each instance, a critical evaluation of the diagnostic and therapeutic procedures is given. This aspect of the book will be particularly useful to the radiologist, who is often called upon to give an opinion as to whether or not surgical treatment is indicated. Particular attention is paid to symptoms indicative of the reversible or irreversible character of lesions. Much space was given to severe bronchial lesions in Boeck's sarcoid.

The book is a credit to Dr. Dijkstra and a welcomed reference work for those engaged in the field of bronchography.

Arch H. Hall, M.D.

Manual of Chest Clinic Practice in Tropical and Sub-Tropical Countries. By A. J. Benatt, M.D., Late Senior Adviser to the World Health Organization. Cloth. Price, $3.00. Pp. 100. E. & S. Livingstone, Ltd., Edinburgh and London; Williams & Wilkins Co., Baltimore, exclusive U. S. agents, 1959.

This compact book, for those interested in the medical and social aspects of tuberculosis, contains some practical and useful information. It consists of two sections and an appendix. The first section covers organization and routine procedure of examination, prevention of tuberculosis, diagnosis and treatment, management of the tuberculous diabetics, and care of the tuberculous pregnant woman and the newborn child. The second section deals with sputum collection and examination for tubercle bacilli, the tuberculin test and BCG vaccination, and the common urine tests. The appendix describes cleaning and sterilizing equipment for tuberculin testing and BCG vaccination and, finally, equipment for a small laboratory.

This book will be of interest primarily to the workers in tuberculosis clinics in tropical and subtropical countries. It should also be of benefit to the residents, nurses and social service workers in tuberculosis sanatoria and clinics; however, for the specialist in tuberculosis, it will not replace the more authoritative textbooks on tuberculosis.

Nathan Levitt, M.D.

Fracture Surgery: A Textbook of Common Fractures. By Henry Milch, M.D., Emeritus Attending and Consulting Orthopedic Surgeon, Hospital for Joint Diseases, New York, and Robert Austin Milch, M.D., Assistant Resident Surgeon, Peter Bent Brigham Hospital, Boston, with a chapter on anesthesia by Herbert D. Dubovsky, M.D., Director of Anesthesiology, Easton Hospital, Easton, Pa. Cloth. Pp. 470, with 671 illustrations. Price, $17.50. Paul B. Hoeber, Inc., 49 East 33rd Street, New York 16, N. Y., 1959.

"This volume is particularly planned for the medical student and surgical house officer, those at the beginning of their study of fractures, as well as the general practitioner who does not devote full time to the treatment of fractures and to whom the fracture is a small and frequently unexpected part of practice." These words of the authors describe an aim for this book, which they have achieved.

The material presented is clearly described and well supplemented with fine drawings and roentgenographic reproductions. The subject is adequately covered for a book of this type. The definitions and explanations and the recommendations for treatment are clearly and concisely stated. The chapters devoted to malalignment, the fundamental principles of reduction of fractures, and the proper use of plaster of

Paris establish a good basis for practical and early consideration of orthopedic problems.

Frank P. Walsh, M.D.

BOOKS RECEIVED

Experimental Nuclear Physics. Volume III. Edited by E. Segrè. Cloth. Price, $23.00. Pp. 811, with many illustrations and tables. John Wiley & Sons, Inc., 440 Fourth Avenue, New York 16, N. Y., 1959.

Neurological Complications of Lymphomas and Leukemias. Publication Number 357 in the American Lecture Series; A Monograph in the Bannerstone Division of American Lectures in Tumors. By Henry M. Williams, M.D.; Henry D. Diamond, M.D., F.A.C.P.; Lloyd F. Craver, M.D., F.A.C.P.; and Herbert Parsons, M.D. Edited by David A. Karnofsky, M.D., Associate Professor of Medicine, Cornell University Medical College, New York, N. Y. Cloth. Price, $5.75. Pp. 134. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1959.

The Year Book of Cancer (1958–1959 Year Book Series). Compiled and edited by Randolph Lee Clark, Jr., B.S., M.D., M.Sc. (Surgery), D.Sc. (Hon.), Director and Surgeon-in-Chief, The University of Texas M. D. Anderson Hospital and Tumor Institute; Professor of Surgery, The University of Texas Postgraduate School of Medicine; Clinical Professor of Surgery, Baylor University College of Medicine; Chairman, Committee on Cancer, American College of Surgeons; F.A.C.S.; and Russell W. Cumley, B.A., M.A., Ph.D., Director of Publications, The University of Texas M. D. Anderson Hospital and Tumor Institute; Professor of Medical Journalism, The University of Texas Postgraduate School of Medicine; Executive Editor, Medical Arts Publishing Foundation. Cloth. Price, $8.00. Pp. 570, with numerous illustrations. Year Book Publishers, Inc., 200 East Illinois Street, Chicago 11, Ill., 1959.

Basic Physics in Radiology. By L. A. W. Kemp, B.Sc., Ph.D., F.Inst.P., Chief Physicist to the London Hospital; and R. Oliver, M.Sc., F.Inst.P., A.M.E.E., Principal Physicist in the Department of Radiotherapy of the United Oxford Hospitals. Cloth. Price, $8.50. Pp. 329, with numerous illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1959.

Intracranial Calcification. By Fermo Mascherpa, Chief of the X-ray Department, Milan Neurological Institute, Milan, Italy; and Vincenzo Valentino, Radiologist of the Institute of Semiological Medicine, Naples University; Neuroradiologist of the Hospital "A Cardarelli," Naples, Italy. Cloth. Price, $9.50. Pp. 150, with 98 illustrations. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Ill., 1959.

Röntgendiagnostik des Magen-Darmkanals. By Prof. Dr. R. Prévôt, and Priv.-Doz. Dr. M. A. Lassrich. Cloth. Price, $28.30. Pp. 346, with 544 illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1959. In the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y.

Dosimetrie und Strahlenschutz; Physikalische und technische Daten. By Prof. Dr. R. G. Jaeger. Leather. Price, $11.80. Pp. 282, with 97 illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1959. In the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y.

Röntgendiagnostik beim Neugeborenen und Säugling. By Dr. Hermann G. Wolf, Assistent der Universitäts-Kinderklinik Wien. Cloth. Price, $23.60. Pp. 317, with 370 illustrations. Wilhelm Maudrich, Franz-Josefs-Kai 23, Wien I., Austria, 1959.

Contemporary Equipment for Work with Radioactive Isotopes. Collected Reports. Translated from Russian. Supplement No. 5 of the Soviet Journal of Atomic Energy, 1958. Paper. Price, $15.00. Pp. 66, with numerous illustrations. Consultants Bureau, Inc., 227 West 17th Street, New York 11, N. Y., 1959.

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Vol. 82, No. 5

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Head

Scott, James H. (Belfast, N. Ireland). Further studies on the growth of the human face. *Proc. Roy. Soc. Med.*, April, 1959, *52*, 263–268.

The author presents a study of bone development of the human face. This is a detailed analysis of embryonic and neonatal growth, well supplemented by diagrams, photographs and photomicrographs. Apparently this report is a supplement to a previous article by the author in 1954.

Growth at Sutures. Each suture presents two "growth centres," one for each boney element. During fetal life the facial sutures are both sites of *growth* and sites of *union* between adjacent bones. With increasing age the function of growth gradually ceases and the function of union becomes consolidated and strengthened.

Cranial Base. Growth of the cribriform plate is complete by two years of age. Union between facial and cranial parts of the ethmoid bone is almost certainly complete by three years of age. Between three and seven years of age the adult relationship between the body of the sphenoid bone, the cribriform plate, the facial ethmoid bone and the frontal bone has been established.

Maxilla. Evidence indicates that over 90 per cent of growth has occurred by the end of the seventh year of life. There is little growth at the circumaxillary sutures during the second decade of childhood.

Growth in Width of Skull. At time of birth, the greater wings of the sphenoid bone unite with the body, and during the first year the two halves of the mandible unite as do the two halves of the frontal bone. After the first year, both the frontal and sphenoid bones are continuous from one temporal fossa to the other.

Alveolar Bone Growth and Tooth Movements. That part of bone which is growing by surface deposition lies beneath the mucoperiosteum of the gum, the deeper layer of which is osteogenetic. The follicles of the developing teeth are attached to the overlying gum. As the bone grows, the developing and erupted teeth are carried in the direction of its growth by virtue of their attachment to the gum.

Relationship of Mandibular Ramus to the Upper Facial Skeleton. The width of the ramus is correlated with the length of the zygomatic arch. Growth of the zygomatic arch is by the process of resorption behind and deposition at the front.

The article is exceptionally complete and detailed. A study of anatomy, such as this, should be of great interest to specialists in this field. An abstract can not do justice to this excellent work.—*David C. Alftine, M.D.*

Stoppa, I. M., and Farinet, G. Malattia di Fahr e suoi rapporti con gli stati ipoparatiroidei; contributo casistico e considerazioni cliniche. (Fahr's disease and its relationship to hypoparathyroidism; presentation of cases and discussion of the clinical aspects.) *Radiol. med.*, April, 1959, *45*, 316–332. (From: Istituto di Radiologia dell'Università di Torino, Torino, Italy.)

Fahr's disease, defined by Fahr himself as "idiopathic non-arteriosclerotic intracerebral vascular calcifications," is characterized pathologically and roentgenologically by bilateral calcifications of the basal nuclei, and clinically by a wide variety of symptoms.

In recent years, this disease has acquired new importance, since it has been frequently observed in patients affected by hypoparathyroidism and since it was thought that the calcific deposits in the basal nuclei were secondary to the altered metabolism of calcium. This hypothesis, however, is not at the moment widely accepted, and the authors, in reviewing the literature on this matter, have found that, while in half of the cases described as Fahr's disease there was associated parathyroid insufficiency, in the other half no hypoparathyroidism was demonstrated but instead neurologic features were present which were related to lesions of the basal nuclei.

The pathologic changes are characteristic: the calcifications are located symmetrically in the caudate, lenticular nuclei and thalamus and in the dentate nucleus. In a few cases the calcific deposits may be localized in the internal capsule, the centrum ovale and in the cortex cerebri or cerebelli. The red nucleus and the subthalamic nucleus (corpus luysi) are never affected by the pathologic process. The histologic findings are of calcium deposits in the periendothelium of the capillaries and in the perivascular spaces of Virchow-Robin in the adventitia and media of the arterioles. Of great importance is the minimal reaction of the nervous tissue surrounding the diseased areas.

Clinically, the disease has been divided into two groups characterized by neurologic disturbances or by features related to the parathyroid insufficiency. In the first group, the neurologic findings are: headache, vertigo, diplopia, hemiplegia or paraplegia, asymmetry of the reflexes, the Babinski sign, involuntary muscular movements, and, when the cerebellum is also involved, ataxia and dysmetria. Other features frequently observed are epilepsy, loss of memory and reduction of the intellectual activity. More varied and complex is the symptomatology presented by patients affected by hypoparathyroidism.

The roentgenologic findings are characterized by symmetric, bilateral calcifications, granular or nodular, or more rarely linear or amorphous, projected in correspondence with the caudate nuclei, lenticular nuclei and thalamus. These calcific deposits are clearly demonstrated in the standard

roentgenologic examination of the skull. Other roentgenologic procedures which are of help in localizing the calcifications more accurately are stereoscopic views and laminagraphy of the skull and pneumoencephalography.

The two cases reported by the authors showed the typical intracranial calcifications localized in the caudate nuclei, and both patients were affected by hypoparathyroidism. Neurologic disturbances related to a lesion of the basal nuclei were not present. On the basis of their cases and of others published in the literature, the authors are of the opinion that Fahr's disease should be considered only in those cases in which there is no evidence of parathyroid insufficiency and in which the main features are those of neurologic abnormalities. All other cases should be considered as secondary manifestations of hypoparathyroidism.—*Antonio F. Govoni, M.D.*

Kuhn, Robert A. Importance of accurate diagnosis by cerebral angiography in cases of "stroke." *J.A.M.A.*, April 18, 1959, *169*, 1867–1875. (Address: 25 Franklin St., Morristown, N. J.)

According to the author the modern treatment of each patient developing a "stroke" rests on the establishment of a precise anatomic diagnosis. The external postures of a stroke are not in themselves at all reliable for the establishment of such an accurate diagnosis. Present day safe cerebral angiography has revolutionized the management of cerebrovascular disease by providing, in many instances, accurate and precise factual information in regard to the cause of the stroke. It is no longer a formidable diagnostic procedure, even for the elderly patient with advanced arteriosclerotic disease.

To support these contentions he presents a series of case reports of patients who were assumed by their family physician to have a perfectly "ordinary" kind of stroke. Angiography in the first patient revealed marked atheromatous narrowing of the proximal portion of the internal carotid artery and surgical reconstruction and anticoagulant medication produced almost complete recovery. In the second patient an arteriovenous anomaly was discovered and removal of this resulted in steady improvement. The third patient was found to have occlusive disease of the left middle cerebral artery and anticoagulant therapy was followed by most satisfactory clinical improvement. A berry aneurysm and blood clot were diagnosed and removed in the fourth patient, with excellent results.

The author feels that the dangers of angiography in cases of stroke are much less than the dangers of delayed or haphazard treatment.—*Arthur E. Childe, M.D.*

Meghrouni, Vahe, and Jacobson, George. The pseudonotch of the atlas. *Radiology*, Feb., 1959, *72*, 260–262. (Address: G. Jacobson, 1200 North State Street, Los Angeles 33, Calif.)

The authors describe a notch-like radiolucent defect in the medial portion of the articular mass of the atlas. This is frequently seen on roentgenograms which are taken of the atlanto-axial joint in the open mouth view projection. It may be mistaken for a fracture or even a dislocation. This pseudonotch defect was reproduced by the use of lead strips on various portions of the superior articular facets of the lateral masses of the atlas. It is formed medially by the margin of the medial tubercle of the lateral mass, which is continuous inferiorly with the inferior articular surface of the inferior articular facet, and laterally by the concave portion of the superior articular facet. Its prominence depends primarily on the dimensions and the shape of the medial tubercle of the atlas.—*W. M. McBride, M.D.*

Neck and Chest

Manfredi, Franco. Atrofia polmonare idiopatica. "Vanishing lung." (Idiopathic pulmonary atrophy. "Vanishing lung.") *Radiol. med.*, April, 1959, *45*, 337–352. (From: Istituto Radiologico degli Ospedali Riuniti, and Ospedale Militare, Trieste, Italy.)

Idiopathic pulmonary atrophy was described by De Martini (1948) as a type of pulmonary emphysema, characterized by an atrophy of the pulmonary parenchyma involving the area of the distal bronchiolar and alveolar structures, and due to a defect in the vascular system of the pulmonary artery.

The author had the opportunity to observe two cases presenting idiopathic pulmonary atrophy and, on the basis of the clinical, roentgenologic, spirometric and bronchospirometric findings, he describes the disease as follows: Idiopathic pulmonary atrophy has generally a unilobar localization and is most frequently asymptomatic. Spirometry and bronchospirometry show a good functional capacity in the affected side due to hyperfunction of the noninvolved lobes. The clinical and roentgenologic findings remain unchanged for many years. Due to the benign evolution of the disease, there are not as yet pathologic and histologic reports to explain its etiology.

The three elements on which a diagnosis can be made are: (1) atrophy of the pulmonary parenchyma; (2) agenesis of the branches of the pulmonary artery in the involved area; and (3) hypoplasia of the distal branches of the bronchial tree in the region of the pulmonary atrophy.

The absence of pulmonary architecture is more marked than in the various types of emphysema; this, in particular, is due to the absence of the vascular markings. The vascular defect, as demonstrated by angiopneumographic studies, involves both the pulmonary artery and vein of the affected area.

Bronchographic studies have demonstrated a progressive and uniform increase in the diameter of the bronchi beginning from the distal portion of the bronchi of the second order. There is no visualization of the fine branches and acini; the distal ends of the dilated bronchi are abruptly interrupted, or show a claw-like appearance or a saccular expansion.

Recently Uehlinger (1958) has united under the designation of "progressive pulmonary dystrophy" both the idiopathic pulmonary atrophy and the vanishing lung. The analysis of the features of vanishing lung, which Burke (1937) defined as a form of bullous emphysema usually involving both lungs, with progressive changes in the size of the cysts, leads the author, however, to conclude that vanishing lung and idiopathic pulmonary atrophy are two distinct pathologic processes with different clinical and roentgenologic findings and courses of evolution.

The two cases are described in detail and the paper is well illustrated.—*Antonio F. Govoni, M.D.*

Khaira, B. S., Young, W. B., and Hart, P. de V. Melioidosis. *Brit. M. J.*, April 11, 1959, *1*, 949–952. (From: General Hospital, Kuala Lumpur, Malayan Fed.)

The authors report 3 cases of melioidosis which were discovered in Malaya. The disease, caused by *Pfeiferella whitmori*, was first reported by Whitmore and Krishnaswami in 1912, from necropsies on vagrants in Rangoon. In 1932, Stanton and Fletcher collected a series of 83 cases of human infection from the literature, in which there were only 2 survivors. In the past the disease has been restricted to that part of Asia east and south-east of India, but in recent years isolated cases have been reported in Great Britain, the United States, and South Africa, occurring in previous residents of the endemic regions.

The mode or source of infection is unknown. The possibility of an insect vector may exist as both the rat flea and the mosquito can harbor and transmit the organism. Direct man-to-man infection has never been reported. At necropsy the lesions are demonstrated in the lungs or bowel. The possibility of ingestion or inhalation of the organism is likely.

The organism stimulates an acute inflammatory reaction and small abscesses are formed. These coalesce and increase in size and eventually break down and discharge, if superficial in location. Spread occurs along deep tissue planes and by blood stream. When bone is involved necrosis results. Sometimes, only the skin may be affected.

The disease may occur in a fulminating form with septicemia, cholera, or severe bronchopneumonia, and with prostration and death within a few days. More frequently it runs a slower but, in the absence of treatment, equally relentless and fatal course of a few weeks or months. Gastrointestinal symptoms may resemble typhoid or dysentery. Pulmonary manifestations are those of lobar pneumonia or lung abscess or may resemble tuberculosis. A severe pyelitis or cystitis may be the presenting form of the disease. A pustular form may be confused superficially with small pox; a benign form with superficial abscesses; and a form with no constitutional symptoms has been described. The acute phase may progress to the subacute or chronic types. All of the cases reported in the white race have presented a subacute or chronic form.

The final diagnosis depends upon the isolation of the causative organism from the pus, blood, urine, cerebrospinal fluid or sputum. Occasionally the diagnosis is made only after guinea-pig inoculation.

Roentgenologically, the findings have differed to some extent in each case. In the first of the authors' cases there were ill defined patchy opacities in the upper zones of both lungs, with a larger homogeneous opacity in the left mid-zone peripherally. In the second case, roentgen-ray examination of the chest was interpreted as showing tuberculous infiltration in the upper and middle zones with cavitation. Roentgenograms of the chest made later, after streptomycin therapy, were interpreted as showing a chronic abscess in the upper lobe of the left lung (probably not tuberculous). In the third case, roentgenography of the chest revealed a loculated hydropneumothorax on the right side. The right upper lobe was partially collapsed and contained a medium-sized cavity surrounded by an irregular area of consolidation.

Grant and Barwell reported the beneficial effects of the sulphonamides. The organisms are sensitive to certain antibiotics. The most generally effective antibiotics seem to be chlortetracycline and chloramphenicol. The combination of the antibiotics and sulphonamides is employed in seriously ill patients.—*J. N. Ané, M.D.*

Gómez Esguerra, Gonzalo; Lichtemberger, Egon; Santamaría, Armando; Carvajal, Lope; Jiménez-Peñuela, Benigno; Saaibí, Edmond; Rey Barrera, A.; Ordúz C., Elio; and Correa-Henao, Alfredo. Familial pulmonary alveolar microlithiasis; four cases from Colombia, S. A. Is microlithiasis also an environmental disease? *Radiology*, April, 1959, *72*, 550–561. (Address: G. Esguerra Gómez, Clínica de Marly, Bogotá, Colombia, S. A.)

The authors present a well documented report of 4 cases of familial pulmonary alveolar microlithiasis from Colombia, South America. The patients were 4 sisters of a family of 7 children in whom the disease was diagnosed at forty, forty-one, forty-six, and fifty years of age.

Pulmonary alveolar microlithiasis is a rare disease, usually familial, which is characterized by the presence of innumerable minute concrements lying free in the alveolar spaces. An interesting and unusual

feature is the complete absence of symptoms. This fact, coupled with the roentgenographic features, makes possible a positive roentgenographic diagnosis even without confirmatory pathologic studies. The etiology is as yet unknown, although it is generally believed that the disease is the result of a disturbance in the respiratory metabolism of carbon dioxide. This would promote alkalinity at the alveolar surface, favoring precipitation of the microliths within the alveoli. The authors are in agreement with this theory but they suggest that environment, diet and living conditions should be considered as additional causal factors. The majority of reported cases shows a familial tendency with two, three or, as reported here, four members of a single family being affected. These patients enjoy many years of apparently good health. Death usually is a result of cardiac insufficiency due to chronic cor pulmonale.

The roentgenographic features are striking and are simulated by no other known disease. There are innumerable shadows of calcific density, about the size of grains of sand, distributed throughout both lung fields. There is a greater opacity of the lower lung fields due to an increased volume of lung tissue, which may be so intense as to efface the cardiac and diaphragmatic outlines. Usually, there are additional linear streaks of density extending from the hili almost to the periphery of the lung, which appear to be calcific deposits along the blood vessels and bronchi.

The 4 cases are presented in detail along with reports of all laboratory and roentgenographic investigations. One patient, aged fifty, expired from chronic cor pulmonale after a symptomatic period of ten years. A report of the postmortem examination is included. The chest roentgenograms and the anatomic and histologic findings, as reported in these cases, are in perfect agreement with other reports of pulmonary alveolar microlithiasis. The authors emphasize that cough is not a regular feature of the disease, and that it appears only when cor pulmonale supervenes, late in the disease.—*Edward B. Best, M.D.*

Bagshawe, K. D., and Brooks, W. D. W. Subacute pulmonary hypertension due to chorionepithelioma. *Lancet*, March 28, 1959, *1*, 653–658. (From: St. Mary's Hospital, London W.2, England.)

Subacute pulmonary hypertension due to carcinomatous metastases to the lung is an established entity. The authors review a case previously reported by McMichael and report 3 additional cases of this entity due to chorionepithelioma.

The important points drawn from these cases are as follows: (a) the disease occurs in women during their menstrual life; (b) it may occur in the apparent absence of a pelvic tumor and in the absence of most of the clinical and roentgenologic changes usually associated with secondary carcinoma of the lungs; (c) the condition, if suspected, is readily recognized by biologic tests for pregnancy; and (d) recent reports of the treatment of chorionic growths are encouraging and make early diagnosis important.

Two of the cases presented were treated with anti-folic-acid and anti-purine drugs and showed good response to therapy.—*George A. Miller, M.D.*

Juliani, Giovanni, Cavallot, Alberto, and Stoppa, Ida. Note sulla fluoroelettrochimografia polmonare; (cinedensigrafia). (Observations on pulmonary fluoroelectrokymography; [cinedensigraphy].) *Radiol. med.*, Feb., 1959, *45*, 132–164. (From: Istituto di Radiologia della Università di Torino, Torino, Italy.)

Twenty normal cases and about 100 cases of pulmonary disease, most of which included primary pulmonary tumors, were examined by means of pulmonary fluoroelectrokymography.

A critical study of the possibilities offered by this method indicates that it does not afford conclusive criteria for the evaluation of the "arteriolar pulsations" and of the "capillary pulse of the lung." In the 49 primary carcinomas of the lung included in this study, the findings were not diagnostic. As a matter of fact, the disappearance of the pulse in the parenchymal thickening and in its vicinity was noted in less than one-half of the cases, while more frequently the findings were normal or nearly normal and the disappearance of the pulse was sharply limited to the region of increased density.

In the cases of peripheral malignancy, benign tumors and non-neoplastic lesions, a disappearance of the pulse which was limited to the involved area was observed.

Graphs referable to transmitted cardiovascular vibrations are demonstrated.

While fluoroelectrokymography cannot afford a definitive diagnosis, it may provide important criteria for the evaluation of the extent of vascular involvement in pulmonary disease.—*Anthony A. Blasi, M.D.*

Livesay, W. R. Clinical and physiologic studies in Ebstein's malformation. *Am. Heart J.*, May, 1959, *57*, 701–711. (From: The South Texas Cardiovascular Clinic and the Cardiorespiratory Laboratory, Hermann Hospital, Houston, Texas.)

Since Ebstein's original description in 1866, 90 cases of this malformation have been reported. An additional case is here presented. The condition is characterized by a downward displacement of the tricuspid valve so that the upper part of the right ventricle is incorporated into the right atrium and functions as part of the atrial cavity. There is an associated atrial septal defect.

Eighty-four per cent of the patients reported in the literature have been cyanotic and a delay (six to twenty-six years) in the development of cyanosis occurred in thirty-seven per cent. The symptomatology is usually mild but many patients do die suddenly, presumably from arrhythmias. The combination of good exercise tolerance with considerable cardiomegaly is significant. Cardiac arrhythmias are frequently present and one-third of reported cases have shown paroxysmal atrial tachycardia, atrial flutter or fibrillation.

The auscultatory finding of greatest significance is a loud third heart sound. The two most commonly encountered electrocardiographic findings are an atypical right bundle branch block and a Wolff-Parkinson-White syndrome.

The significant roentgen features are globular cardiac enlargement with relatively small vascular pedicle and diminished pulmonary vascularity. Fluoroscopically, pulsations are diminished in all dimensions except for the area of the pulmonary conus where they are more vigorous. Angiocardiography reveals a large, dilated right atrium with stasis of the contrast medium in this chamber. The pulmonary vasculature is poorly outlined. Soloff and others have demonstrated a narrow, non-opacified band in the most anterior portion of the heart, in the right anterior oblique position. This is thought to represent the displaced tricuspid leaflet.

The most important finding at cardiac catheterization is intracardiac right to left shunt. Pressure tracings reveal atrial pressure curves across the entire precordium to the left cardiac border. Only along the uppermost portion are ventricular pressure curves recorded. The finding of normal right ventricular and pulmonary artery pressure is almost diagnostic in a cyanotic patient.

The primary physiologic defect is ineffective right atrial emptying, leading to marked reduction in pulmonary blood flow. The resulting right atrial dilatation and increased blood volume ultimately lead to an increase in pressure and right to left shunt with accompanying cyanosis in the presence of an atrial septal defect or patent foramen ovale.

The differential diagnosis considers pulmonary stenosis with or without septal defect, tricuspid atresia and tetralogy of Fallot.—*David Morse, Jr. M.D.*

Espino-Vela, J. Rheumatic heart disease associated with atrial septal defect: Clinical and pathologic study of 12 cases of Lutembacher's syndrome. *Am. Heart J.*, Feb., 1959, *57*, 185–202. (From: Department of Congenital Heart Diseases, National Institute of Cardiology, Mexico, D. F.)

The author reports 12 proved cases of mitral valvular damage associated with wide atrial septal defect, 11 by autopsy and one by operation. In contrast to the classical Lutembacher's syndrome, these cases showed double mitral lesions and rheumatic involvement of other valves; hence, the author's designation of rheumatic heart disease associated with atrial septal defect.

The relative rarity of this disease is emphasized considering that atrial septal defect is the second most frequent congenital cardiac malformation in Mexico City, and that cases of rheumatic heart disease make up over 40 per cent of the admissions to the National Institute of Cardiology in Mexico. In each case myocardial rheumatic valvular disease was severe and extensive, quite often affecting more than one valve. This ruled out the congenital nature of the mitral stenosis.

The clinical features suggestive of association of rheumatic valvular disease and atrial septal defect were: (1) atypical auscultatory findings of rheumatic mitral stenosis, absence of presystolic accentuation of the diastolic rumble; (2) the presence, quite commonly, of a systolic murmur accompanied by a palpable thrill in the pulmonary area; and (3) cyanosis of slight to moderate degree (not seen in the absence of heart failure).

The supportive roentgenologic findings of this association were: (1) the large size of the pulmonary main trunk and its branches; (2) vigorous pulsations; (3) a mitral or mitral-tricuspid cardiac contour; and (4) slight or absent left atrial enlargement.

Electrocardiographic data suggestive of this disease were: (1) the presence of right bundle branch block, and (2) commonly, qR type complexes in Lead V_1 with (3) concomitant left atrial enlargement ("mitral" P waves).

Pathologically, the right atrium was the largest chamber. The left atrium varied in size from slightly enlarged to normal. The atrial septal defects seemed to be the result of extensive resorption of the septum primum with faulty development of the septum secundum. In 3 of the 12 cases reported, there was virtually a single atrium. The pulmonary arteries were widely dilated, but lung sections from 5 cases did not reveal important vascular arteriolar damage.—*J. T. Ling, M.D.*

Johnson, John L. Postpericardiotomy syndrome in congenital heart deformities. *Am. Heart J.*, May, 1959, *57*, 643–653. (From: The Department of Pediatrics, College of Medical Evangelists, Los Angeles, Calif.)

The postpericardiotomy syndrome occurs in patients in whom the pericardium has been opened during surgery for congenital heart conditions. The major feature of the syndrome is a pericarditis, sometimes associated with a pleural effusion or pneumonia. Recognition of the condition is important as treatment is different from that in postoperative in-

fectious infiltrations of the lung and other causes of sudden cardiac enlargement. The syndrome is usually observed between one and two weeks postoperatively and is found in various age groups.

Eleven cases are reported. The syndrome occurred in 3 cases of pulmonary stenosis, 7 cases of atrial septal defect (2 of which were associated with transposed pulmonary veins and one with mitral insufficiency) and in one case of aortic stenosis. Hypothermia was used in all but 2 patients. Symptoms usually developed in less than seven days or between fifteen and thirty days. The most frequently encountered symptoms were fever and chest, shoulder or arm pain. The most common physical findings were poor heart tones, friction rub, decreased breath sounds, dullness of the chest, and tender or enlarged liver. Roentgenograms revealed heart enlargement either at the time of these signs and symptoms or one to two days thereafter. Also noted in several cases were decreased pulsations of the heart on fluoroscopy, pleural effusion and pneumonia. Treatment consisted of bed rest and digitalis when indicated. The 3 patients who received antibiotics made no more rapid a recovery than those who did not. Only one patient in this series did not completely recover and this case was complicated by pulmonary infarction, subacute bacterial endocarditis and probably a number of small coronary infarctions. There is no evidence of rheumatic activity and the etiology is unknown.—*David Morse, Jr., M.D.*

Davis, W. H., Jordaan, F. R., and Snyman, H. W. Persistent left superior vena cava draining into the left atrium, as an isolated anomaly. *Am. Heart J.*, April, 1959, *57*, 616–622. (From: The Cardiac Unit, Department of Internal Medicine, University of Pretoria, Pretoria, South Africa.)

A case report is presented giving symptoms, differential diagnosis and treatment of the very rare persistent and patent left superior vena cava. The vena cava emptied into the left atrium, as was demonstrated by cardiac catheterization from the left arm. The persistent left vena cava was the only congenital defect present in the patient. The condition was remedied when the abnormal vein was tied off.—*J. H. Harris, M.D.*

Beahrs, Oliver H., and Schmidt, Herbert W. Dysphagia caused by hypertrophic changes in the cervical spine; report of two cases. *Ann. Surg.*, Feb., 1959, *149*, 297–299. (From: Mayo Clinic and Mayo Foundation, Rochester, Minn.)

Slight to moderate hypertrophic changes of the cervical spine are not uncommon after the age of fifty. These changes may cause some degree of local or radicular pain in various positions of the head and neck. It is unusual, however, for them to become large enough to produce severe pain or difficulty in swallowing because of pressure on surrounding nerve structures, the hypopharynx, or the cervical esophagus. Such a possibility should be borne in mind when dysphagia is persistent and when organic lesions in the hypopharynx or cervical esophagus have been excluded.

Two such cases are reported. In both cases the chief complaint, clinical findings, and roentgenographic manifestations were almost identical. An anterior hypertrophic osteophyte of moderate size was found between the fourth and fifth cervical vertebrae. In each instance the dysphagia disappeared after surgical excision of the hypertrophic spur.—*J. T. Ling, M.D.*

Keller, A. Paul, Jr. Some considerations concerning chicken bones in the esophagus. *South. M. J.*, April, 1959, *52*, 414–418. (From: Department of Otology & Rhinolaryngology, Emory University School of Medicine, Emory, Ga.)

In the past ten to fifteen years chicken bones lodged in the esophagus have become of increasing interest due, in part, to the very rapid growth of the chicken industry. The patient is most often in the age group over forty years and very frequently wears upper dentures, which prevent feeling the bone until it has passed the palate.

Chicken bones which lodge in the esophagus are usually situated in the cricoid region. They are rarely demonstrated in posteroanterior roentgenograms but they are shown in lateral views of the neck. Although they may appear spicular in the roentgenogram they are usually proven to be flat when removed.

The author feels that if the plain lateral roentgenogram of the neck fails to show a foreign body and considerable doubt still exists as to its presence, it may be better to proceed with an esophagoscopy rather than to administer barium.—*Arthur E. Childe, M.D.*

Skeletal System

Jensen, Wallace N., and Lasser, Elliott C. Urticaria pigmentosa associated with widespread sclerosis of the spongiosa of bone. *Radiology*, Dec., 1958, *71*, 826–832. (Address: E. C. Lasser, Presbyterian Hospital, Pittsburgh 13, Pa.)

It must be considered that the disease urticaria pigmentosa exists not only as a localized pathologic process characterized by an intradermal collection of mast cells, but also as a widespread pathologic process which may involve many systems. The authors present the seventh case to be reported in the English

literature of urticaria pigmentosa with generalized osseous and abdominal visceral involvement. The generalized osseous process is a diffuse osteosclerosis as a result of the replacement of cancellous bone by an increase in the number and thickness of the trabeculae. In the case presented, this finding was well demonstrated, particularly in the metacarpal and the phalangeal bones of the hands. Generalized osteosclerosis of this type is uncommon in diseases other than myelosclerosis and fluorine poisoning. Cases of systemic urticaria pigmentosa have been reported which exhibit localized skeletal defects. However, these defects in the bones failed to show a common pattern and had not been biopsied. Therefore, only the cases with the generalized form of osseous involvement should be considered representative at present of systemic urticaria pigmentosa.—*W. M. McBride, M.D.*

Bose, K. S. Osteo-articular lesions in smallpox. *J. Indian M. A.*, Aug. 16, 1958, *31*, 151–154. (From: Department of Orthopaedics, Medical College Hospitals, Calcutta, India.)

The incidence of osteoarticular lesions following variola is small—less than 0.5 per cent. Children are usually affected during the pustular stage or later during the desquamation of the scabs. The larger joints are involved and the patient develops pain, swelling and fever. Two clinical varieties are noted: (1) a suppurative type leading to abscess formation, osteomyelitis and arthritis probably due to secondary infection; and (2) a nonsuppurative type characterized by mild clinical features and roentgenologic evidence of bone necrosis of the epiphysis and metaphysis causing epiphyseal damage with resultant development of later deformities.

Twelve patients are reported—all children. The roentgenographic appearance resembled chronic pyogenic osteomyelitis but differed from it in that the ends of the bones forming the joints were affected. Small cystic changes and subperiosteal bone formation without sequestration and without marked necrosis and new bone formation were characteristic. Both the epiphysis and the metaphysis were involved along with the joints. In the late phases of the disease, the joint space was diminished and the articular ends were deformed. Motion was somewhat restricted.—*Frank J. Rigos, M.D.*

Blood and Lymph System

Richards, L. Stephen, and Thal, Alan P. Phasic dye injection control system for coronary arteriography in the human. *Surg., Gynec. & Obst.*, Dec., 1958, *107*, 739–743. (From: Department of Surgery, University of Minnesota Medical School, Minneapolis, Minn.)

A phasic dye injection control apparatus for injection of opaque media into the aorta for visualization of the coronary arteries is described and the results are illustrated. The device consists of 2 units: a compressed nitrogen-driven syringe; and an electronic dye injection control system triggered by the R wave of the electrocardiogram, thus allowing injection at variable times in the cardiac cycle. Injection times can be varied from .2 to 1.6 seconds. With a large bore catheter (P.E. 330), 65 cc. of 90 per cent hypaque can be injected in from .6 to 1 second with 150 pounds pressure per sq. inch. In 10 dogs intra-aortic pressures were recorded with a second end-opening catheter placed with the tip 1 cm. from the end of the injection catheter. A 15 to 35 mm. of mercury increase in intra-aortic pressure was noted during injection. The increase in pressure was rapidly dissipated by the expansile aorta. There have been no findings in dog or human that have been attributable to deleterious effects of injection pressure. In 11 patients on whom coronary arteriography has been performed with this device, the procedure has proved to be entirely safe.

In actual practice, only 30 to 50 cc. was injected, depending on the heart rate and patient's age.—*Lois Cowan Collins, M.D.*

Massell, Theodore B., Heringman, E. Craig, and Greenstone, S. M. An evaluation of newer opacification media; diatrizoate and diprotrizoate. *A.M.A. Arch. Surg.*, Feb., 1959, *78*, 293–299. (Address: T. B. Massell, 6221 Wilshire Blvd., Los Angeles, Calif.)

The authors report on the results obtained and the effects produced in 308 cases of vascular opacification when the following media were used: diatrizoate sodium (hypaque sodium), sodium and methylglucamine diatrizoate (hypaque M or renografin), and sodium diprotrizoate (sodium miokon).

Mild systemic reactions occurred in 2.9 per cent of the cases and no serious reactions were noted. When the mixed diatrizoate salts were injected into smaller veins or arteries, severe pain was rarely produced and it was never persistent; in some cases, there was moderate pain. However, on occasion, severe pain in the small vessels was produced when either of the pure sodium salt preparations was administered. Of the two, the diprotrizoate was found to be more irritating than the diatrizoate.

The opacification of the peripheral vessels was excellent when any of these three media was employed. However, consistently good aortic opacification was achieved only with the mixed diatrizoate salts. The authors recommend that the use of the more toxic vascular opacification media such as acetrizoate and iodopyracet be abandoned in favor of the diatrizoate solution. Specifically, they prefer the sodium and methylglucamine diatrizoate.—*James A. Martin, M.D.*

Savage, C. R. Retrograde aortography in acute dissecting aneurysm of the aorta. *Lancet*, Feb. 7, 1959, *1*, 281–282. (From: Warwick Hospital, Warwick, England.)

The diagnosis of acute dissecting aneurysm of the aorta can often be made by clinical findings alone or assisted, on occasion, by roentgenography of the chest. The use of contrast media is seldom indicated but may be of value in the diagnosis of the doubtful case, since the presence and extent of the dissection and the degree of obstruction of the aorta and its branches can be shown. Aortography may be of greater value when dissection involves the iliac arteries. Percutaneous retrograde femoral catheterization results in slight inconvenience or risk even to a seriously ill patient.

The author reports a case of acute aortic dissection with obstruction of the right iliac artery. He illustrates the method used and shows how surgical management might have been modified, perhaps with successful outcome, had the significance of the roentgenographic findings been appreciated at the time. The fifty-three year old male patient died of uremia five days after a second operation.

The author, in retrospect, expresses the opinion that the demonstration by aortography of bilateral renal artery obstruction should have made obligatory a decompression fenestration procedure on the aneurysm in the thoracic aorta, and that this should have been supplemented by a further decompression of the aneurysm in the right iliac artery. The importance of detecting obstruction of the renal arteries is emphasized and it is suggested that retrograde aortography can be an effective help in deciding treatment.—*Roscoe E. Miller, M.D.*

Pérez Anguiano, Mannuel. La aortografía como medio diagnóstico en urología. (Aortography as a diagnostic procedure in urology.) *Rev. urol., México*, Nov.–Dec., 1958, *16*, 257–273. (From: Hospital Civil, Guadalajara, México.)

Aortograms for visualization of the aorta and renal vessels have been found useful in the diagnosis of pathologic conditions of the kidney and vessels and in other conditions such as hydronephrosis and chronic infection. The translumbar approach was used in most cases, introducing the needle into the left lumbar region. Catheterization of the femoral artery or one of its branches was also utilized at times. A combination of both methods has produced the best results. The contrast media used in the cases presented were neo-iopax and renografin.

The procedure, which at first seems radical, does not have many complications. Most frequently encountered in this series was extravasation of the opaque medium with more or less pain at the site of the injection. One case showed symptoms of shock with profuse sweating. In another, pain radiated into the left side and arm. One case in which the left renal artery was inadvertently injected developed no complications.—*Murray M. Friedman, M.D.*

Foster, John H., and Killen, Duncan A. Aortography and peripheral arteriography: An analysis of results in a University Medical Center. *Ann. Surg.*, March, 1959, *149*, 321–341. (From: The Department of Surgery, Vanderbilt University School of Medicine, Vanderbilt University Hospital and Thayer Veterans Administration Hospital, Nashville, Tenn.)

The authors reviewed 233 aortograms and peripheral angiograms made from 1950 through 1957 at the Vanderbilt University School of Medicine with emphasis on failures and complications.

Of the 25 thoracic aortograms 18 (72 per cent) were considered diagnostic. Most of the thoracic aortograms were obtained through a catheter inserted via the brachial, subclavian or femoral arteries. One transventricular injection was also made. Urokon 70 per cent or hypaque 50 per cent was used. The injection was done manually and rapid serial exposures were made. Two carotid injection failures were due to extravasation of the contrast medium. Four brachial artery catheter injections failed because of inadequate contrast.

Of 127 abdominal aortograms 89 (70 per cent) were found to be diagnostic. Local anesthesia was used in 35 per cent of the cases, spinal anesthesia in 50 per cent and general anesthesia in 15 per cent. The usual puncture at about L1 was used, but two needles were employed in many cases. Twenty-five cc. of urokon 70 per cent was injected and a series of eight rapid short (0.03 to 0.1 sec.) exposures were made. In 25 per cent of the cases a single exposure was made. There were 2 major complications and 12 minor ones. There was one death which could not be directly attributed to the procedure and one permanent paraplegia following extravasation of the contrast medium at D10.

Of 81 peripheral arteriograms (77 femoral, 3 popliteal and 1 brachial) 61 proved diagnostic. Local anesthesia was used and 15 cc. of urokon 30 per cent or hypaque 50 per cent was injected. Two major complications occurred requiring above the knee amputation in each case due to gangrene following popliteal artery thrombosis.

A detailed discussion of injection and roentgenographic techniques, anesthesia, complications, equipment and contrast media is given along with 26 recommendations which should help in obtaining the best results from the procedures.—*Frank M. Windrow, M.D.*

Botseas, D. S., and Lawrence, G. H. Skin necrosis complicating femoral arteriography. *Ann. Surg.*, Jan., 1959, *149*, 135–139. (From: Department of Surgery, Mason Clinic and the Virginia Mason Hospital, Seattle, Wash.)

The authors report 2 cases of necrosis of areas of skin of the thigh following femoral arteriography. They reviewed 2 other similar cases previously reported. The first patient was a sixty-six year old

diabetic who developed a 4×8 cm. bullous area following femoral arteriography and bilateral sympathectomy. This enlarged somewhat and progressed in about eight to ten days to a complete area of necrosis. This was excised and grafted.

A second patient developed two similar areas following femoral arteriography and the insertion of an aortic graft. These ulcers were very painful. Following excision and grafting, this patient also remains well.

Microscopically, the involved skin area showed a necrotic ulcer with other changes of skin infarction. The reason for this complication is not clear, but probably results from a large bolus of contrast material entering a small arterial branch.—*George L. Sackett, Jr., M.D.*

Panke, William F., Bradley, Edward G., Moreno, Augusto H., Ruzicka, Francis F., Jr., and Rousselot, Louis M. Technique, hazards, and usefulness of percutaneous splenic portography. *J.A.M.A.*, March 7, 1959, *169*, 1032–1037. (Address: L. M. Rousselot, 170 W. 12th St., New York 11, N. Y.)

This is a report in considerable detail of the techniques that the authors use for percutaneous splenic portography.

Under local or general anesthesia, 50 cc. of sodium acetrizoate is injected into the spleen through an 18 gauge spinal needle. Multiple films are exposed at intervals up to thirty-two seconds.

Pain in the left upper quadrant frequently occurs following this procedure, but it is usually transitory. Bleeding requiring transfusion was observed in 2 per cent of 172 cases. Laparotomy was not necessary in these cases. Infection occurred in 2 per cent as did false puncture into the peritoneal cavity, kidney, and liver.—*George L. Sackett, Jr., M.D.*

Piguet, B., and Écoiffier, J. Maladie hémolytique (type Minkowski-Chauffard) et malformations veineuses congénitales. (Hemolytic disease of Minkowski-Chauffard type and congenital venous malformations.) *Presse méd.*, Feb. 25, 1959, *67*, 381–382. (From: Hôpital Cochin, Paris, France.)

Excellent phlebograms of an eighteen year old young woman (diagnosed to have congenital hemolytic anemia at the age of six and treated by splenectomy at the age of eight are presented to support the authors' thesis that abnormal and dilated veins, especially of the lower extremities, are part of the picture of congenital familial hemolytic disease. This could be an important factor in the pathogenesis of chronic ulcers of the lower extremities of such patients. The authors believe that stasis in these dilated and abnormal veins (appearing as a bluish patch at the medial aspect of the left leg) could have eventually led to ulceration in spite of splenectomy ten years earlier. In fact, a localized periosteal reactive thickening could be demonstrated along the fibula at the same level. Frequently various other congenital osseous and ocular deformities are reported associated with this disease.—*Jirair N. Sarian, M.D.*

Buzzi, Alfredo, Fustinoni, Osvaldo, Gutiérrez, Thomás J., and Bur, Juan B. Aneurisma bacteriano (micótico) de las arterias radial y tibial posterior. (Bacterial aneurysm (mycotic) of the radial and posterior tibial artery.) *Prensa méd. argent.*, Sept. 12, 1958, *45*, 3029–3032. (Address: A. Buzzi, Pueyrredón 615, Buenos Aires, Argentina.)

The authors report a case of a thirty-seven year old woman who was admitted for observation because of a tumor in the middle third of the left leg. The mass was about the size of a lemon and showed expansile pulsations. The skin over the lesion was tense, shiny and showed a few superficial veins. Palpation caused no pain. Prior to the appearance of the tumor, the patient had localized pain in this area. Another pulsating tumor about the size of a plum was found in the middle third of the left forearm. Eight years prior to admission, the patient had an attack of polyarticular rheumatic fever. Three years before admission she had anginal attacks with pain which radiated into both arms.

Physical examination revealed an aortic stenosis and insufficiency. Arteriograms were made by injecting the femoral artery in Scarpa's triangle using a 50 per cent contraxine. Roentgenograms made two seconds after the injection showed the aneurysm outlined and its relationship to the posterior tibial artery. In the third and fourth roentgenograms, the aneurysm remained opacified, but the radiopaque material had cleared from the arteries of the leg.

Both aneurysms were resected. The aneurysm in the radial artery showed evidence of thrombosis and calcification. Septic emboli and bacterial endocarditis are found in 90 per cent of the cases which develop mycotic aneurysms; however, no evidence of endocarditis was found in this case, suggesting that the infection was a transient phenomenon.

Most of the aneurysms appear at sites where the arteries are not protected by skeletal muscles and are subject to trauma, such as in the popliteal artery, the femoral artery in the inguinal region, and in the brachial artery. Arteriograms provide a method by which the aneurysm can be located with reasonable certainty and also provide information concerning the collateral circulation.—*Murray M. Friedman, M.D.*

General

Campbell, Crawford J., Roach, John F., and Jabbur, Munir. Xeroroentgenography; an evaluation of its use in diseases of the bone

and joints of the extremities. *J. Bone & Joint Surg.*, March, 1959, *41-A*, 271–277. (From: The Departments of Orthopaedic Surgery and Radiology, Albany Medical College, Albany, N. Y.)

Xeroroentgenography is the recording of roentgen images with a standard roentgenographic camera by a photoelectric process rather than the photochemical one used in conventional roentgenography. An electrostatic image of the size, shape and radiodensity of an interposed object is produced on a xeroplate, which is a metallic plate coated with a semiconductor such as selenium. The image is made visible by placing finely divided powder granules on the charged pattern of the xeroplate. The processing unit is small and can be moved easily. This method requires no roentgenographic film or darkroom, and the whole procedure usually takes less than fifty seconds.

The method has particular application in examination of the extremities, and permanent records can be made by photographing the xeroplate image on 35 mm. film. One advantage is that fine detail of both bone and soft tissues is obtained on the one xeroplate image. A disadvantage is in the considerably higher mas. required. Because of this, the heavier parts of the body, such as the skull, abdomen, pelvis and lumbar spine, were not studied in this evaluation of the method. Reproductions of standard roentgenograms, xeroroentgenograms, and photographs of a pathologic specimen are included.—*Douglas S. Kellogg, M.D.*

Donnan, M. G. F. Torulosis. *J. Fac. Radiologists*, Jan., 1959, *10*, 17–20. (From: Royal Perth Hospital, Perth, Western Australia.)

The author reports 5 cases of torulosis which demonstrate the diverse manner of presentation of the disease and indicate the difficulty in diagnosis.

A short historical review is given. The first clinical description was made by Busse in 1894 and further considered by Buschke in 1895.

The organism is *Cryptococcus neoformans*. Its modus operandi is a single cell, which reproduces by budding and, in the tissues, is surrounded by a clear zone of gelatinous material. Histologically, the miliary nodule is the most common pathologic lesion. Where there is a paucity of organisms, the lesion may be a fibrotic granuloma. Where there is massive proliferation of *Cryptococci*, the lesion may be a gelatinous mass. The port of entry may be the skin, and the intestinal or respiratory tract, the last being most frequent. An interesting note is that 10 to 30 per cent of patients with torulosis have associated neoplasia of the reticuloendothelial system.

The best known manifestation of torulosis is a large solitary nodule in the lung, and cure has been reported after removal of such lung lesions. Involvement of the brain and meninges is uniformly fatal. The bone lesions present only a sclerosis very similar to an osteitis.

In the cases presented, the course seemed prolonged and the symptoms confusing. One case was asymptomatic and was only studied when a lung lesion was noted on mass chest survey.

Awareness of the possibility of a mass lesion in the lung being torulosis may spare the radiologist the humiliation of finding the diagnosis post mortem.—*Eva S. Carey, M.D.*

Doyle, Owen W. Unusual gas-forming infections. *Radiology*, Jan., 1959, *72*, 94–96. (Address: 1013 Professional Village, Greensboro, N. C.)

Abnormal gas shadows in any part of the body should suggest the possibility of a gas-forming infection. Although gas gangrene is usually derived from the clostridial bacteria, the anaerobic streptococci, the diphtheria bacillus and *Actinomyces* may also produce similar manifestations. These organisms thrive in necrotic tissue and usually spread along muscle planes.

The author reports 2 cases of this type. In the first case gas produced by *Clostridium welchii* was noted in the abdominal wall, the psoas muscle, the terminal ileum, and the right colon. These gas pockets simulated free air in the peritoneal cavity and a laparotomy was done for a perforated viscus. At this time a leukemic mass involving the cecum was found producing an area of necrotic tissue which served as a focus for the gas bacillus infection. The second case was that of a diabetic male suffering from a rectal abscess. Roentgenograms of the lower abdomen and scrotum showed soft tissue swelling and a large amount of gas in the scrotum. Anaerobic streptococci were found to be the cause of this gas-forming infection.—*John Alexander, M.D.*

Sepúlveda, Bernardo, Jinich, Horacio, Bassols, Francisco, and Muñoz, Rafael. Amebiasis of the liver; diagnosis, prognosis, and treatment. *Am. J. Digest. Dis.*, Jan., 1959, *4*, 43–64. (From: Department of Gastroenterology, Department of Radiology, and Department of Surgery, Hospital de Enfermedades de la Nutrición, México, D. F.)

It is estimated that in Mexico amebiasis affects from 15 to 30 per cent of the total adult population. The authors present a study of 80 cases of hepatic involvement representing about 3 per cent of all cases of amebiasis seen by them in a Mexico hospital.

Diagnosis of this lesion depends primarily upon signs of right upper abdominal sepsis, especially in view of the fact that only 22 per cent of the cases had dysentery and in only 25 per cent was *E. histolytica* recoverable from the stools. Roentgenographic find-

ings were minimal in cases of amebic hepatitis with varying degrees of right diaphragmatic elevation and limitation of motion. However, in cases of amebic abscess these changes were more marked, often associated with basal atelectasis and different degrees of pleural effusion. On occasions, diaphragmatic humps appeared over abscesses in the dome of the liver and less frequently air-fluid levels could be seen within abscess cavities. Roentgenographic studies were of considerable aid in the diagnosis of the pulmonary complications of empyema, hepato-bronchial fistula and pulmonary abscess. These pulmonary complications occurred in approximately 1 out of each 6 of their cases.

The proper treatment depends upon the severity of the disease but in all instances must include the use of emetine or chloroquine. When abscess drainage is required, this can be done either by trocar aspiration or surgical drainage, being mindful in either case of the seriousness of pleural or peritoneal contamination.—*James A. Martin, M.D.*

Meyers, Paul T. Joint changes in swine erysipelas. *Radiology*, Jan., 1959, *72*, 86–93. (Address: The Gilfillan Clinic, Bloomfield, Iowa.)

Erysipelothrix rhusiopathiae suis is an organism which invades the circulatory system and parenchymal tissue of the swine where it may produce septicemia, dermatitis, and arthritis. Fowls, birds, fish, and man may also be afflicted. Methods of diagnosing this condition in swine were studied by a team of research veterinarians of the United States Department of Agriculture and used by the author. Culture of the organism was found to be expensive and difficult. Hematologic and cutaneous tests have not been perfected. The author observed that the most rapid and least costly method of diagnosis is roentgenographic examination of the infected joints. The outstanding roentgenologic feature of the disease process is new bone formation, especially along the anterior or dorsal margins of the carpal and tarsal articulations. This is observed in mild cases as small bony spurs or bony plateaus and in advanced cases as fused anterior joint margins. The soft tissue is invaded by a continuous proliferation of bone which has the appearance of a cauliflower type of expanding growth. The underdevelopment of the bones in the joints is obvious and indicates that normal bone growth and development have been inhibited during the early weeks of the swine's life. The sole method of preventing this disease is vaccination of the newborn pig.

In man the disease is confined to the handlers of raw pork or fish infested with these organisms. The affected person develops erysipeloid skin lesions on the hands and forearms. The rash may also involve the skin on the body. These lesions are usually amenable to treatment with the administration of penicillin or sulfa to the infested individual.—*Wallace M. McBride, M.D.*

Deibert, Kirk R. Radiographic examination of surgically removed specimens with a radiologic-pathologic correlation. *Radiology*, Nov., 1958, *71*, 723–728. (Address: Box 22, Rt. 1, Jackson Highway, Florence, Ala.)

Roentgenograms of gross surgical and autopsy specimens will frequently reveal an interesting and instructive radiologic-pathologic correlation with the preoperative studies. Frequently they will give information that cannot be obtained by black and white photography, and may act as a convenient and useful substitute if photographic facilities are not available. The technique of obtaining such roentgenograms is described.

Some of the conditions which may well be demonstrated by this technique include calcified cardiac and vascular lesions, pulmonary lesions containing calcium, solid and cystic tumors of the kidney and adrenal glands which frequently contain calcium, fallopian tubes which may show some calcification due to tuberculous infection or extrauterine pregnancy, uterine myomas, lesions of the gallbladder, polypoid tumors of the gastrointestinal tract, bone lesions, and other miscellaneous lesions, particularly when calcium is present.—*Walter H. Jarvis, Jr., M.D.*